ANNALS OF SURGERY

VOL. 103 MAY, 1936 No. 5

TRANSACTIONS
OF THE
SOUTHERN SURGICAL ASSOCIATION
MEETING HELD IN HOT SPRINGS, VA.

PRESIDENTIAL ADDRESS

THE MASTER SURGEON AND THE LAYMAN

ROBERT LEE PAYNE, M.D.
NORFOLK, VA.

ROBERT BROWNING wrote: "Man's reach must exceed his grasp, else what's a Heaven for?" Thomas Carlyle stated that "Our main business is not to see what lies dimly at a distance, but to do what lies clearly at hand."

If there is an apparent inconsistency here between the thought of the poet and that of the historian, as the two apply to our problem, it is only an apparent inconsistency. Surely, we of this Association and of similar and kindred Associations are not to be content to reach only for such objects as we can certainly attain, or we are but little more than craftsmen. But surely, too, we are to do what lies clearly at hand, or we are but dreamers. If we are only dreamers, then we are not exemplars of Kelvin's proposition that the life and soul of science are in its practical application.

We are here today as practical men. Scientists, we ought to be; idealists, we hope we are; but practical men we must be, if we are to play our full part in the joint effort that brings us here together.

Because we are scientists, and because for program purposes this address had to have a title, I have captioned this address, in part, "The Master Surgeon," and because we are practical men, I have called it, in other part, "and the Layman." The two, in their actual existence, touch each other so intimately as to be well-nigh indivisible. Let us consider first, separating him for the moment, The Master Surgeon. What is he? What are his qualifications?

Passing as a matter of course, as well established these days, the requisite that he possess the breadth of education and general knowledge that is attested by an academic degree or adequate academic credits, we demand also, as a matter of course, that he be thoroughly and firmly imbued with knowledge of the principles of medicine and surgery as inculcated by approved medical schools. After he has obtained his license to practice medicine, with or without the experience of internship, he is legally qualified—legally, be it noted—to practice surgery. He is not fitted, however, to practice surgery. He is not, indeed, well fitted to practice medicine. So much the less is he fitted to practice surgery, without supervision.

Let us assume that he has become an adequately equipped medical man. And he must be that first. There is, I fear, a tendency today on the part of many young graduates to assume that they can safely leap from the medical school, over all experience of the practice of medicine, into the field of surgery. That assumption is not justified. The practical surgeon who is in truth a Master Surgeon knows that Mayo and Archibald, and all others, are eternally right when they say that the surgeon must be primarily a well trained practitioner of medicine, and he holds with Haggard, and the others who have said, that the scientist must never become so interested in the disease that he forgets the patient. Let us say, then, that the aspirant has become an adequately equipped medical man. Either he is now prepared to be trained and to train himself to be a Master Surgeon, or he has been under training to that end while he was equipping himself as a medical man after his graduation. For the teaching and the learning of surgery are as surely postgraduate work as is the earning of the academic degree of Ph.D. a matter of postgraduate work. The principles of surgery, to repeat, are taught in the medical schools. The application of these principles is learned after the medical schools have conferred their degree of M.D.

I stress the difference—and not as a grammarian—between the words "taught" and "learned," because in the medical schools the aspiring surgeon is taught by those who are—certainly as to the student—primarily teachers. That is literally true of teachers who are full time men. It is also virtually true—as to the students—of those part time men, surgeons in active practice, who instruct classes or groups. Both classifications *teach* the principles of surgery. But the application of these principles must be *learned* by the aspirant. And it can best be learned from, rather than taught by, Master Surgeons who are not primarily teachers.

At least, that is the case in the main today. Eventually, the system under which the University of Minnesota grants first, the degree of Bachelor of Medicine, and later, after compliance with specified requirements, the degree of Doctor of Medicine, may be so extended and developed as to include a degree of Doctor of Surgery. The holder of such a degree may have been required to serve so rigid an apprenticeship, in the most favorable circumstances, in the practical application of surgical principles, as to have become a qualified surgeon. That would mean, however, that he must have under-

gone a period of three or five or even eight years of postgraduate application. Such a degree based upon such requirements is at this time, however, among those ideals for which we may reach but which we cannot as yet grasp.

There remains, then, the utilization of such resources, such facilities, such advantages, such opportunities, for the production of Master Surgeons as we have.

In a wholly inadequate manner, we attempt such utilization in the awarding of the F.A.C.S. degree by the American College of Surgeons. This degree, I think we all know, merely represents fulfilment on the part of its holder of requirements which are far too lax and which are, in a number of respects, of doubtful wisdom. Indeed, it represents something unpleasantly like a membership in a trade union. Worse, it represents membership in a trade union which does not obligate itself—as does so well conducted a labor organization as the Printer's Union, for example, to produce a sufficient supply of properly trained members. In this regard, the American College of Surgeons does those things which it ought not to do, and leaves undone those things which it ought to do. And yet, the F.A.C.S. is the only, even remote, approach to that degree of Doctor of Surgery, or Master of Surgery, toward which we are somewhat gropingly reaching.

Permit me to digress and say that this reaching for a Master's qualifications is as much, if not more, for the benefit of the layman than for individual attainment. The layman has no guide by which he may choose a qualified surgeon, and while our first duty is to provide adequate postgraduate teaching, it is equally important that we educate the layman, and provide a reliable criterion by which he may know to whom he may entrust his life or that of his family. For the time being, this latter responsibility must be put squarely upon the shoulders of the hospitals. Only qualified Master Surgeons should be given full privileges of the operating rooms in our hospitals, and this should be the very foundation upon which the American College of Surgeons is built. By this criterion only may our layman be educated to choose and select. By this means, when our layman is hurriedly urged into the hospital for surgical attention, he will quickly learn that his family practitioner, whom he idolizes, is not always competent to handle the problem which is to be faced.

In an article in the ANNALS OF SURGERY for October, 1935, which is the product of an exceptionally extensive survey and illuminating analysis, Doctor Archibald points out that the Royal Australasian College of Surgeons requires, among other things in the creation of its Fellows, "a minimum of five years' postgraduate training in surgery under an approved senior surgeon."

As there are many men, there are many minds, so it is easily conceivable that three or four years of postgraduate training of this character would suffice to equip some men, while others might need six or eight. Nevertheless, as fully as I realize the weaknesses and the dangers of standardization, I am not inclined to dispute the conclusion that there should be a minimum of five years of such training.

Such a requirement might work a hardship in some cases, but the compelling need of providing the greatest good for the greatest number should guide. There is much wisdom in the axiom of our brothers of the Bar that "hard cases make bad law." What they mean is that the disinclination to work a hardship leads to the abandonment of inexorable logic. It will not do to lower our standards in order to avoid working a hardship here and there. Neither the medical schools nor our Associations, neither public spirited Master Surgeons nor informed laymen can make capable surgeons, still less great surgeons, but together they can prevent incompetent men calling themselves surgeons, from visiting their incompetence upon the ignorant and suffering public.

That the public does suffer by the thousands from such men, is common knowledge among us. That it suffers in this manner because it is ignorant, is equally common knowledge among us. But it is not the public that is to be blamed for its ignorance in this respect; nor is the blame attributable merely to an inscrutable and immutable scheme of life. "The fault, dear Brutus, is not in our stars, but in ourselves . . ." For our own sake, for the sake of what has been called "the most brilliant of arts," we must, then, seek to repair this fault, as well as for the sake of the layman who is, or ought to be, our partner in this effort.

So far as law is concerned, it is repeated, any man who holds the degree of Doctor of Medicine and has a license to practice medicine may operate upon and practice surgery upon the layman. Before a lawyer may appear before the Supreme Court of the United States, he must have been licensed for three years to practice before the highest court of his own state. But the young doctor who has had his license to practice medicine for one day may take the body of an unfortunate patient in his hands, and with unskilled scalpel send its soul winging to the last of all Courts.

It is submitted that it is our duty so to inform the layman, to educate him, if we like, that he will not tolerate such a condition. It is our duty to inform him that a considerable percentage of the work done by experienced and capable surgeons is work of repair undertaken for the purpose of saving limbs, organs and often lives desperately endangered by the clumsy and sometimes culpable ignorance of incompetent men.

It is our duty to inform him that one of the most potent causes of blundering surgery is the open hospital, where any doctor may operate who can induce a sufferer to trust himself to his inexperienced hands. It is our duty to inform him that the cause, the foundation itself, of the open hospital, in turn, is the necessity in present conditions that presses upon the hospital's management to keep its beds filled. It is our duty to inform him that the shingle of an M.D. is not at all an assurance that the man in the office before which it swings is capable of practicing surgery—whether he be young, mature, aging, or aged.

If he asks—this layman—how he is to know what doctor is capable of practicing surgery upon him, it behooves us to answer, for the present, that

only such surgeons as are approved by senior surgeons, or by the management of closed hospitals, are to be entrusted with the complicated diagnostic and technical tasks that must be performed by the competent surgeon. It behooves us to tell him, too, that it is for him and his fellow layman to join with us in our effort, our purpose, to see to it that eventually only qualified men shall be legally authorized to practice surgery.

Our layman, being a business man, a man of affairs, may say: "It is all very well to insist that your graduate shall devote so many years to postgraduate study and training and experience, that he will not be permitted to practice surgery without supervision until he is well past the age of 30 years. But who is to support him during those postgraduate years? Why should he be denied the young man's right to marry and rear a family?"

Our answer, I take it, will be: "As our medical schools are constituted now, and as postgraduate work is carried on, the aspiring surgeon must earn, must be helped to earn, a living while he is assistant to a Master Surgeon." After we have grasped a larger measure of that for which we reach, after that superschool of surgery of which we now dream shall have been established, and shall have become prepared to train Master Surgeons, then our layman and his associates must create and maintain foundations whose pecuniary yield will enable the wise and experienced men who conduct this superschool to finance selected men of intellectual distinction, of strong will and of high character, while they pursue their quest of the Master Surgeon's scroll.

Our layman being a man of affairs, who has made his own way, who is familiar with the vigor and energy and courage of youth, may also ask: "Can you, in this idealistic plan of yours, be right in urging that studious and learned, intellectual and even brilliant, academically taught, and scientifically trained men, shall have withheld from them the right to practice surgery alone as Master Surgeons until the first fine flavor of youth has departed from them?"

Our answer, in my judgment, should be: "Yes!" Knowledge is not wisdom. Brilliance is not experience. Courage is not judgment. This is concretely answered by Disraeli when he said: "However gradual may be the growth of confidence, that of credit requires still more time to arrive at maturity."

Knowing how to operate is not knowing when to operate. Diagnostic acumen does not necessarily take into account other factors than the difficulties before the surgeon—factors which must be taken into account by the Master Surgeon. The caution which must go hand in hand with intrepidity, the comprehension which must range far beyond the operative field, the sort of fearlessness that is not afraid of being thought afraid, the wisdom that makes knowledge useful—all these are less the property of fine flavored youth than of full bodied maturity. And all are essential items of the Master Surgeon's equipment.

If by taking earnest thought and by converting that thought into careful

action we can form the Master Surgeon by such a mold as I have tried to suggest, and if the layman in his turn can be brought to realize that only such a Master Surgeon should be authorized to practice the science and art of surgery, then finally we shall have grasped that for which we have been reaching, and the practice of surgery will have become distinctively the province of the Master Surgeon to the incalculable benefit of the layman.

Finally, as tonight represents the crowning episode of my incumbency as your President, it behooves me to say that what little work I have done has been a labor of love. The glamour of high office does not appeal to me, and I would remind those who follow me that the dizzy heights are cold, the valleys warm and cheerful. I would that I had words to thank you again for the great honor conferred upon me.

The iris of the summer's shower, the rosy dawn, the brilliant hues of the bird of Juno are bright, soft things—but blend them, blend all the beauties of Nature in one harmonious whole, and there will still be wanting that mysterious essence of gladness that enters into the heart of man when so richly rewarded by his fellow man. Of all honors greatly to be desired, this to me is the greatest.

In closing, may I ask your forgiveness for a personal allusion. My father, my grandfather, and my great grandfather were all doctors of medicine. As a young boy my interest in medicine carried me into the library of my grandfathers, and there on the inside page of their old volumes I saw written, in the handwriting of my fathers, the following words: *"Esse quam videri."* Freely translated this means: "To be, rather than to seem to be." To attain to the meaning of this motto has been an obsession of mine since I first began the study of medicine.

Success in the practice of surgery is not a wild dream of technical attainment, nor a madness of desire for professional acclaim. Oh! no, it is not that. It is goodness and honor and sincerity of effort and faith in life. It is this that I am wishing for you and yours.

BRAIN ABSCESS

JOSEPH E. J. KING, M.S., M.D.
NEW YORK, N. Y.

IN THIS paper I shall confine my remarks for the most part to the *treatment* of brain abscesses secondary to infection of the middle ear, mastoid and accessory nasal sinuses, as well as those of traumatic origin. The subject of metastatic brain abscess will not be considered. The etiology, pathology and diagnosis of brain abscess have been adequately covered by Macewen,[21] Eagleton,[14] Evans,[15] Atkinson,[2] Adson and Craig[1] and various other writers.

Otogenic abscesses resulting from otitis media and mastoiditis are in the majority, and extension of infection from these sources most frequently involves the temporosphenoidal lobe and the cerebellum. Evans[15] states that 83.2 per cent of brain abscesses are due to spread of infection from otitis media and mastoiditis, and that 9.1 per cent result from infections of the nasal cavity and its accessory sinuses. Only 7.7 per cent arise from all other sources. Therefore, 93.2 per cent of all brain abscesses are actually due to extension of infection from the nasopharynx. He further states that the spread of infection to the brain substance from the middle ear and mastoiditis, by *direct extension*, is through purulent thrombosis of the lateral sinus, osteomyelitis of the tympanic wall, along the adventitial spaces of perforating blood vessels, and through the internal auditory meatus. The spread of infection from the nasal cavity and its accessory sinuses by direct extension is due to osteomyelitis of the sinus wall, along the perineural spaces, and along and through the veins. In addition, the involvement of the brain by direct extension follows trauma of the skull and invasion of the brain by malignant growths.

Regardless of the mode of infection, according to Evans, a brain abscess commences with an inflammatory leukocytic infiltration and softening of the brain substance, and terminates by the conversion of a portion of the brain into a cavity containing pus. In the *acute* form, the abscess has no definite wall. It consists of a broken-down area of brain tissue, which might be termed acute suppurative encephalitis, with a central area containing pus and brain detritus. The wall, if it exists at all, is irregular and ill defined and consists of brain tissue with a shaggy surface, surrounded by an area of edematous brain substance. Should the patient be fortunate, this process becomes *chronic* in that a well defined wall or "capsule" is established, and the abscess then truly follows the old surgical description of any abscess as being "a circumscribed cavity containing pus" (Fig. 1). The wall of the abscess consists of an internal layer composed of purulent tissue, the so called pyogenic membrane, and an external layer of granulation tissue composed of various proportions of fibrous macroglia and all fibrous tissue derived from the vascular adventitia and, in some instances, from the leptomeninges. The granulation tissue is infiltrated with plasma cells, lymphocytes, and a few eosinophile

leukocytes. The infiltration is continued in the adventitia of the vessels for a considerable distance into the brain (Evans). Bagley,[3] in 1922, described the formation and pathology of a brain abscess and its wall.

It has not been determined accurately how long it takes for a definite wall or capsule to form. Penfield[23] stated that the wall of an abscess begins to form in the first week, but that it does not offer resistance to a brain cannula till after two or three weeks. Grant[17] stated that a definite capsule or firm wall is

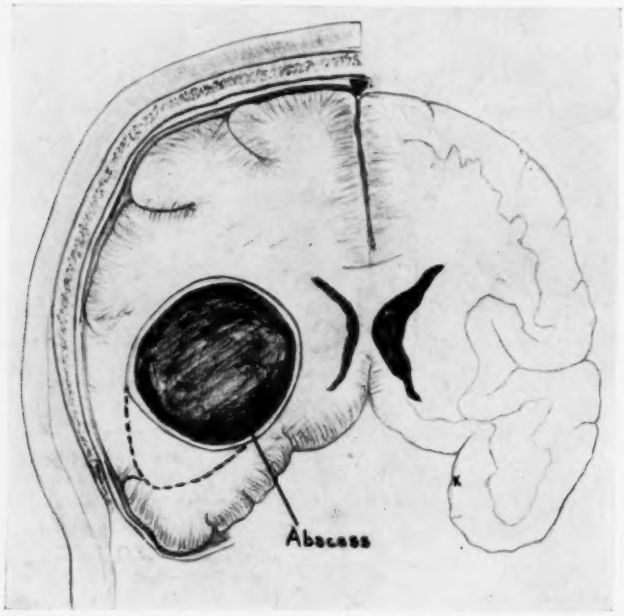


FIG. 1.—Chronic encapsulated abscess of the temporal lobe. The hemisphere which contains the abscess is larger than its mate. The ventricles are markedly compressed. The adjacent ventricle may be completely collapsed. The structures are shifted to the opposite side. The broken line indicates that the proximal pole may extend downward and outward and may form a definite "stalk" fixed to the dura.

well established in the interval between the fourth and sixth weeks, and Bagley showed a section of an abscess wall which formed in three to four weeks. The latter also stated that the thickness and firmness of the abscess wall will be determined by the relative amount of mesoblastic and gliablastic fibrous tissue present, the amount of glial tissue in the wall, and the length of time the abscess has existed.

PRELIMINARY MANAGEMENT.—When a brain abscess is suspected, *stereoroentgenographic* examination should be made in order to determine the degree of involvement of the mastoids, ethmoids, frontal sinuses, sphenoidal cells and antra. This may throw considerable light on the neurologic picture. Should infection of the sphenoid coexist with the involvement of the ethmoids and the frontal sinus, and a frontal lobe abscess is properly treated, a subsequent unsuspected abscess in the temporosphenoidal lobe due to sphenoidal infection may develop. The lack of attention paid by many writers to the

eradication of the source or focus of the infection in the middle ear, mastoid, or the nasal accessory sinuses is rather remarkable. Except in a case of emergency, the infectious process or processes in the sites of *extracranial* origin should first be eliminated before the intracranial suppurative process is attacked. Otherwise, following the proper handling of a subcortical abscess, another abscess may develop from the same source. Furthermore, an extradural lesion which may simulate a subdural one may be eliminated without a subdural exploration. In most instances of brain abscess, the drainage of the abscess can be accomplished immediately following the extracranial procedure. In some critical, comatose cases accompanied by marked slowing of the pulse, it may be advisable to evacuate the abscess first and perform the extracranial operation at a later date.

TYPES OF OPERATION.—In a general way the types of operative procedures which have been described may be classified as follows: (1) The closed method. (2) The open method. (3) Extirpation of the abscess *in toto*. (4) Tapping.

Closed Method.—In the closed method, usually a small opening is made in the skull. Some type of drainage tube, *e.g.*, a small rubber tube or a small rubber catheter, is inserted into the abscess cavity without inspection of the cavity, the tube is allowed to remain in place for a considerable period of time, and is slowly removed without any, or but a small, degree of herniation. Coleman,[8, 9] Grant,[17] Dowman,[13] Sachs,[24] Bagley, McKenzie,[22] Atkinson, Davidoff[12] and others have advocated this method with some few changes or special details in the technic being recommended by them. In this method one first locates the "capsule" of the abscess and introduces the drainage material, catheter or tube, into the abscess cavity without intentional leakage of pus into the surrounding brain substance. For this purpose various ingenious devices in the form of guides, directors, encephaloscopes and searchers have been used. Among these is the encephaloscope devised by Whiting, its modification described by Eagleton,[14] and the cannula, sleeve and tube by Grant. Both Grant and Coleman have reported rather large series of recoveries following operation by the closed method. Macewen's use of the decalcified chicken bone drain is well known.

It is difficult to understand how an abscess with secondary extensions, loculations, or of an irregular shape could be properly drained by this method. Moreover, it is believed that the capsule, in the cases of so called recoveries, collapses about the drain as it is slowly removed, leaving a partially obliterated tract with a thick fibrous wall which remains as a potential source of infection. Such an instance was observed in 1919 after an abscess had been drained in this manner. Deep or centrally situated abscesses, however, can be drained only by this method, or by the use of a cannula or searcher.

Open Method.—In 1924,[19] the author advocated a rather wide open approach to the abscess, removal of the overlying cortex and a portion of the presenting capsule, evacuation and irrigation of the abscess cavity, thorough inspection of the cavity, and eventual obliteration of the cavity by its tem-

porary eversion and herniation. This method was advocated following the recovery of a patient in 1920 and of two other patients in 1923 who were operated upon in this manner. Previous to this time drainage tubes had not proven satisfactory for one reason or another, and in the method described they were completely discarded. The principles set forth at that time are still adhered to in general, with some modifications which will be dealt with later in this paper.

In 1925, Cahill[4] reported a series of cases treated by the use of the Mosher drain which was first considered by Mosher in 1915. The abscess was operated upon in two stages and was entered, in the majority of cases, by the mastoid route. Subsequently, in papers presented in 1929 and 1933, Cahill[5, 6] advocated a more extensive removal of the brain cortex and capsule of the abscess, so that the drain could be more accurately placed into the abscess cavity. He stated that "the formation of a fistulous tract about the drain to the abscess cavity in part makes the capsule extend to the surface of the brain," which approaches in some degree the method described by the author. In a personal communication, Cahill[7] described the formation of a small hernia or "nubbin" at the site from which the drain was finally removed in the cases operated on according to his modified method. It is believed that this small hernia represents the remnants of the contracted abscess cavity which has found its way to the surface. Cahill described the use of the Mosher drain in cases of otitic abscess and advocated its use in cases treated by the otologic surgeon.

Horrax,[18] in 1934, described a procedure first advocated by Dr. Cushing 10 or 12 years earlier for certain chronic encapsulated brain abscesses. He termed it "marsupialization." It decidedly belongs to the open method. Horrax made an opening in the skull about the size of a half dollar, a similar opening in the dura, removed the overlying cortex, removed a portion of the presenting abscess wall after partial evacuation of the abscess, fixed the cut edges of the capsule to the galea with sutures, and filled the abscess cavity with gauze lightly packed over a thin layer of gutta percha. Over this packing of gauze another layer of perforated gutta percha was placed and the entire area was covered with dry dressing and bandage. Subsequently the gauze and gutta percha were gradually extruded by herniation of the abscess cavity outward and were finally removed. A hernia cerebri may have developed, but this subsided. The area became covered with granulation and epithelialized. In this procedure thorough removal of the contents of the abscess cavity and inspection of the cavity can be accomplished. With the exception of suturing the edges of the capsule to the galea, this procedure, in general principles, is quite similar to the one proposed by the author. Horrax reports only one death in a series of eight cases operated upon by him according to the marsupialization method. Sachs reported a successful case operated upon by this method.

In 1932, the author[20] operated upon a case of temporosphenoidal abscess which had spontaneously ruptured through the mastoid wound. The capsule

of the abscess was almost completely empty, was contracted to about the size of a malaga grape, and was surrounded by a considerable area of necrotic and suppurative brain substance. In this instance the pericapsular necrotic brain was removed by suction, and inasmuch as the small contracted capsule could not be utilized in any manner, it was removed. The central portion of two layers of iodoform gauze was inserted into the resulting excavation in the temporosphenoidal lobe, and strips of soft iodoform gauze were packed into the cavity against the two layers of iodoform gauze, in the manner of a Mickulicz tampon, until the entire excavation was gently but snugly filled. Dakinization of the wound was carried out and the superficial dressings were changed daily after the first 48 hours. The iodoform gauze packing was slowly pushed out by the gradual herniation of the excavated brain surface and a portion of it was removed each day until none remained. The two layers of iodoform gauze were carefully removed from the brain surface which had herniated to the level of the skull. The area granulated over and healed. Later excision of the scar with approximation of the scalp edges was accomplished.

Since this time iodoform gauze has been used in a similar manner both in encapsulated and non-encapsulated abscesses, and in one instance in which there was a considerable amount of pericapsular brain suppuration and necrosis similar to the case described above.

Adson and Craig, in 1935, described a procedure somewhat similar to those advocated by Horrax and the author. They made an opening in the skull about 3 cm. in diameter, sutured the meninges and the cortex together, to prevent separation of the cortex from the dura, opened the dura by a crucial incision, so as to expose an area of cortex about 2 cm. in diameter, and then sealed the meninges to the cortex by electrocoagulation. After aspiration of pus from the abscess cavity, followed by gentle irrigation with saline solution, the capsule of the abscess was opened with the electrosurgical needle. This allowed of inspection of the abscess cavity aided by an illuminated retractor. Two rubber tubes, sections of a No. 18 F. catheter, were inserted into the cavity and about these strips of iodoform gauze were loosely packed. These strips of gauze were shortened and removed by the tenth day, leaving the two tubes in position. One of these tubes was shortened every other day, while the other one was removed gradually as the sinus closed in and forced it out. The time required from four to six weeks. They stated that there was but one death in 15 consecutive cases of supratentorial abscesses and one death in three cases of cerebellar abscesses. One may judge from the description of their procedure that the approach to, and inspection of, the abscess cavity classify it as an open method, while the use of drainage tubes with the formation of a sinus might place it in the category of the closed method.

It may be deduced that the operative procedures proposed or devised by Cahill, Horrax, Adson and Craig and the author have many similarities in principles though they may differ in details of technic.

Extirpation.—Various writers have reported an occasional case in which an old chronic abscess has been completely extirpated. Most of these were probably removed after an osteoplastic flap had been turned down in exploration for a tumor. Adson and Cairns report such instances. I am not acquainted with anyone who advocates routine extirpation of diagnosed abscesses, although one of the French surgeons stated that he did. When the abscess is of long standing, has a very thick wall, is calcified or contains thick inspissated pus, extirpation without opening the abscess might be considered. Or in a case of spontaneous evacuation of an abscess cavity, which is surrounded by suppurative necrotic brain tissue (as in the case which I have reported above), extirpation would then have to be done for the reason that it cannot be utilized.

In the instances of large abscesses which closely approach the ventricle, extirpation would surely be followed either by rupture into the ventricle, or rupture from the ventricle outward due to the increasing intraventricular pressure. I feel that the major and inner portion of the capsule should be preserved as a barrier and serve to prevent such a catastrophe.

Tapping.—Dandy,[10, 11] in 1926, and again in 1932, advocated tapping only in cases of chronic abscesses. He introduced a ventricular needle through a small trephine opening and a tiny nick in the dura into the abscess cavity and left the needle in place until the pus ceased to drip. He neither aspirated nor irrigated the cavity, but withdrew the needle and closed the scalp incision tight. He stated that "many times a single release of pus has been necessary; on fewer occasions two or more taps have been required."

I have had no experience with this method. The fact that it is not used by many authors who have reported a fairly large number of recoveries would seem to indicate that tapping is not generally used. Recently I saw sections of three brains which had been removed from patients who had been operated upon consecutively by one of my colleagues according to this method. He told me of the autopsy findings in a fourth case. He has given up the use of this type of operation. In passing, it might be said that the use of the osteoplastic flap in connection with the operation for brain abscess in general is not used and should not be advised.

It will thus be seen that there is wide divergence of opinion regarding the proper approach to, and the method of drainage of, an abscess of the brain, although most writers agree that the operation should be done after the abscess has developed a substantial wall or capsule. It is believed that no single procedure or combination of operations will result in 100 per cent recoveries. All abscesses are not alike; therefore, no one procedure will suffice in all cases.

SURGICAL TECHNIC.—*Anesthesia.*—Local anesthesia, novocain 1 or ½ per cent solution with suprarenin alone or combined with avertin, is preferred. With adults, especially those who are stuporous or semicomatose, local anesthesia alone will suffice. With children the combination of avertin and novocain is more satisfactory. The use of ether is not advised for the

reason that it increases intracranial pressure while avertin decreases it. Vomiting following ether administration may produce damage to the abscess wall or result in rupture of the abscess into the ventricle. Ether also increases bleeding.

Procedure.—The vast majority of all cerebral abscesses in my experience have approached the dura at a point where it can be attacked at a depth which varies from ½ to about 3 cm. Inspection of museum specimens shows similar findings. Therefore, the approach to the abscess, as advocated in my original paper, with the modifications in technic to be described, will allow of adequate drainage of the majority of cerebral abscesses, whether the source of infection originates from the mastoid region and middle ear, the nasal cavity and accessory sinuses, or trauma. As stated above, the source of the intracranial infection should be eliminated before operation for the abscess, except in traumatic and in certain critical or extremely grave cases.

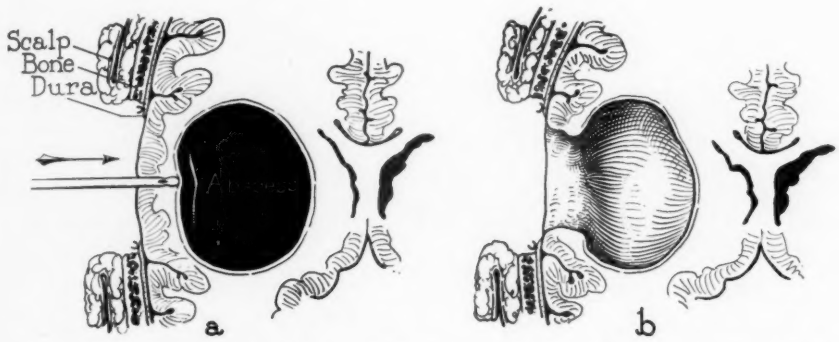


FIG. 2.—(a)—A cranial and a dural opening have been made directly over the abscess. The dural margins are fixed to the meninges and cortex by electrocoagulation and interrupted fine catgut sutures. The cannula inserted into the brain substance meets with resistance not unlike that of a thin rubber ball. (b)—The overlying cortex and a portion of the wall of the abscess have been removed. The cavity can be inspected thoroughly. Fixation of the dura to the meninges and cortex prevents spreading of infection into the meninges.

In a case of temporosphenoidal lobe abscess, a short, straight, slightly oblique incision about one and one-half inches is made through all the soft parts down to the outer table of the skull. An opening with a bone drill is made at a point about one inch posterior to and about one and one-half inches above the external auditory meatus. A small self-retaining retractor gives adequate exposure and controls bleeding. A small nick is made in the dura and a specially devised blunt cannula of small caliber is introduced and passed directly inward and slightly downward. When the end of the cannula comes in contact with the wall or capsule of the abscess (Fig. 2-a), resistance similar to that offered by a thin-walled rubber ball is encountered. It is preferred that the cannula should not enter the abscess cavity. It is not necessary, and spreading of infection is obviated while preparation for drainage of the abscess is being made. When the wall of the abscess cavity is located, slow tangential insertions of the cannula will help to ascertain the location and approximate size. By means of the cannula it can be determined whether the abscess is situated more anterior, superior, posterior or inferior to the

opening in the skull. When the position of the abscess is determined, the mastoid incision is extended upward over the ear at a distance of about half an inch from its attachment. The oblique straight incision is connected with it and the three scalp flaps thus defined are dissected up from the pericranium and held with self-retaining retractors. The anterior flap carries the ear downward and forward out of the way. This exposes an area of pericranium about 3 or 4 cm. in diameter depending upon the size of the abscess. The bony opening in the skull is enlarged in a circular manner to about the size of a silver dollar and in the direction necessary to place it directly *over* the abscess. The pericranium is removed from the outer table of the skull only sufficiently to permit making the proper sized opening in the bone, and is not stripped off from the outer table of the skull for any distance beyond the bony opening. In some cases where the abscess extends low into the temporosphenoidal lobe it may be necessary to remove the bone downward into the mastoid wound. The dura is incised in stellate manner to within about one-quarter of an inch of the bony margin of the defect and the dural flaps are excised. This leaves a circular opening in the dura slightly smaller than the bony opening into which the cortex bulges. The cortex is fixed to the dura by means of interrupted sutures of fine plain catgut as suggested by Adson, and the dural margin, meninges and cortex are fused by means of electrocoagulation. The three narrow strips of iodoform gauze which were formerly inserted beneath the dura, between it and the cortex, to prevent spread of infection into the subdural space, are no longer used in this manner but are placed in a tangential position about the bony opening to cover over the edges of the bone and dura. The subsequent oozing of blood into this gauze followed by clotting of the blood fixes the gauze to the bony and dural margins and forms a mechanical barrier against the spread of infection. The overlying brain substance is removed by means of the electrosurgical unit and suction to expose an area of the wall of the abscess about the size of a quarter or a two-franc piece, depending upon the size of the abscess. Should pericapsular necrotic and suppurative brain substance be present it can readily be removed by suction. This necrotic brain material if encountered is usually external to that portion of the capsule which is exposed, but it may extend laterally about the capsule. As the overlying brain substance is removed, the capsule comes into view and presents itself like the end of an egg. An aspirating needle is inserted into it and a small amount of the pus is removed in order to lessen the intracapsular tension and is preserved for laboratory examination. The capsule is then opened with the electrosurgical needle and the pus is removed by suction. The outer presenting portion of the capsule is next removed in a circular manner so that the complete interior of the abscess cavity can be inspected (Fig. 2-b). Should an opening into an extension of the abscess be found, a sufficient portion of the capsule is removed to convert both into one cavity. After the pus has been removed and the cavity cleaned the inner wall will appear rather smooth, but may have thin flakes of inspissated pus loosely attached to it. These can

be easily removed under direct inspection. By this time it will be observed that the depth of the cavity is not so great as when the cavity was first entered. The floor of the cavity will have progressed upward toward the operator or outward toward the opening in the skull. This lessening in depth varies from about one-quarter to one-half inch in the cases which have been observed. This elevation or outward progression of the floor of the abscess after the outer portion of the capsule has been removed is due to release of pressure from within the abscess, followed by increase in the size of the compressed ventricles.

The central portion of two layers of iodoform gauze, about six inches square, is introduced into the abscess cavity (Fig. 3-c), and strips of *soft* iodoform gauze are gently but rather snugly packed against the two layers of iodoform gauze so that the two layers of gauze come in intimate contact with the wall of the entire cavity (Fig. 3-d). The packing is continued until the

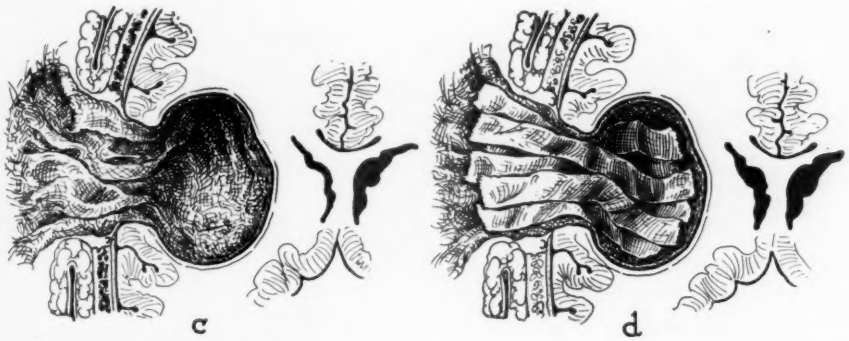


FIG. 3.—(c)—The central portion of a pack consisting of two layers of iodoform gauze have been inserted into the abscess cavity and the free edges have been brought out through the openings in the dura and skull. (d)—Narrow strips of soft iodoform gauze have been gently packed into the soft pack so as to bring the two layers of gauze into opposition with the inner walls of the cavity and the tract through the cortex.

tract is completely filled to the level of the skull, and the loose margins are folded over the packing. The self-retaining retractors are removed, bleeding points are controlled by electrocoagulation or suture ligature if necessary, and fluffed iodoform gauze is loosely stuffed beneath the flaps. The open end of a small rubber irrigating tube is carried within the overlying gauze, and a wet azochloramid gauze dressing is applied and held with a bandage which includes the lower jaw so as to prevent displacement of the dressing.

AFTER TREATMENT.—*Dressings.*—The first dressing is done after 48 hours. All the dressing down to the incision is removed and replaced. Meanwhile instillation of azochloramid solution into the superficial dressing in the wound is made every two hours. (Azochloramid solution is used now in place of Dakin solution for the reason that it is more prolonged in action, does not irritate the scalp so much as Dakin solution, and is readily obtainable and easily prepared.) On the third day the iodoform gauze packings in the external portion of the wound beneath the flaps are loosened and wet with the solution. This allows of slight progression outward of the iodoform

gauze in the brain abscess cavity proper. At daily dressings the narrow gauze strips are gradually removed from the operative wound and from within the iodoform packing lining the abscess cavity (Fig. 4-e). On the fifth day the iodoform gauze strips are removed from about the bony margin. By the tenth day the floor of the abscess cavity will have become elevated sufficiently to extrude and allow complete removal of the gauze within the cavity. The last two layers of iodoform gauze are slowly and carefully removed. While doing this it is well to use a rubber ear syringe with a soft tip and hydrogen peroxide. Where the wall is but weakly encapsulated the gauze will have become firmly adherent to the floor of the abscess, and must be very slowly and carefully removed, otherwise the surface of the brain will be torn. This is not the case when the abscess is well encapsulated.

Rapid herniation of the abscess cavity is no longer advised nor permitted. It is necessary only to allow the floor of the abscess to reach the level of the

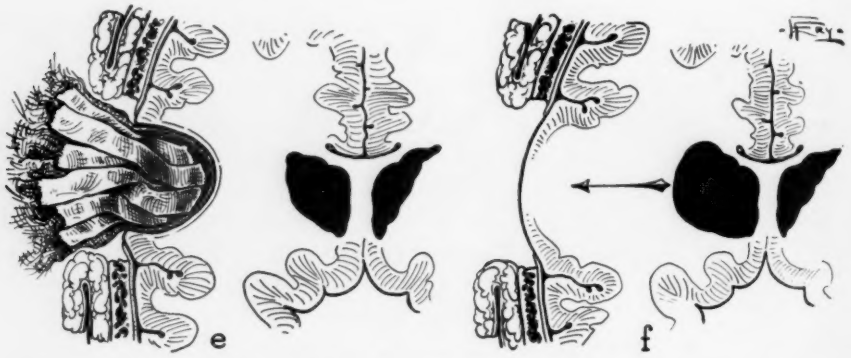


FIG. 4.—(e)—The gauze strips have been slowly shortened at successive dressings accompanying slow and gradual elevation of the floor of the cavity. This controlled herniation outward is due to accumulation of fluid in the ventricles with slow removal of the gauze. (f)—The floor of the cavity is maintained at the level of the skull by lumbar punctures, should they be necessary. The remnants of the abscess wall become a part of the scar tissue at the operative site. The margins of the bony defect become covered with granulation tissue and the scalp flaps are allowed to fall over and become adherent to the area. Development of an actual hernia cerebri is *not* allowed.

skull or near the level of the skull (Fig. 4-f). This slower outward progression of the floor of the abscess which reaches completion by the tenth day is controlled by lumbar punctures done as often as is necessary. It is also due in a considerable degree to the fact that the gauze is maintained in position in the cavity. Iodoform gauze for this purpose has proved to be of greater value than the rubber dam formerly used. Meanwhile, the scalp flaps have been allowed to fall back into position and become attached to the underlying tissues.

The iodoform gauze which was placed in the mastoid wound is removed on the sixth day and thereafter changed every other day. In the case where a mastoidectomy has been done simultaneously with evacuation of the abscess, the removal of the squamous portion of the temporal bone between the site of the operation for the abscess and the mastoid process is usually done, for the reason that this bone is frequently involved. The dura in this region is

kept intact. Since overherniation or development of a hernia cerebri at the site of operation is not allowed, the difficulty formerly offered by the overhanging hernia into the mastoid wound does not exist. The area now becomes rapidly covered with healthy granulations followed by epithelialization. In order to permit more rapid spread of epithelium across the surface, the scalp about the wound is kept well shaved and the granulating area is covered with strips of perforated adhesive placed across from one scalp flap to another. After the area has been covered with adhesive strips, irrigation of the wound is discontinued and a dressing of wet azochloramid gauze is applied.

In this type of abscess the patient should be out of bed after about 16 days, should be able to leave the hospital about the end of the third week, and the wound should be completely covered with epithelium within four or five weeks. After about six months to a year I have usually performed a scalp plastic in order to cover the area more securely with normal scalp. This affords better protection and prevents excoriation of the scar, especially in summer. Most of the scar following the plastic procedure is in the hairline, and that portion behind the ear over the mastoid region is rendered less conspicuous.

Usually the postoperative temperature reaches its highest point, about 101.5° or 102° F., on the first or second day postoperative, and after a few days recedes to normal.

Lumbar Punctures.—Lumbar punctures can safely be done on the third or fourth postoperative day, and thereafter as often as is necessary to prevent elevation of the floor of the abscess beyond the level of the skull. When the abscess is very large it may be necessary to resort to a lumbar puncture daily. In one instance where the abscess cavity contained more than seven ounces of pus and in which later ventricular distention was rapid and considerable, daily lumbar puncture was done with removal of from one and one-half to two ounces of fluid. Removal of too much fluid will produce a negative pressure in the ventricle with resultant headache, while removal of too small an amount will result in bulging of the floor of the cavity. It should be remembered that recession of the floor continues to some extent after removal of the fluid; therefore the needle should be withdrawn when the floor of the cavity is at the level of the skull. Headache will thus be obviated.

General Care.—The head of the patient is kept elevated, at first on one or two pillows. On the third day the patient is placed in a semisitting position. Fluids are readily taken after the patient has recovered from the anesthetic. It has not been necessary to give tubal feeding, and intravenous administration of glucose is not necessary. It is important that the bowels should move well daily. An enema is given every morning, and a mild saline laxative or cathartic at night should this be necessary. When the bowels have not moved well, it will be noticed that the brain surface tends to bulge, while the reverse is true if the bowels have been properly evacuated. Usually the patient complains of very little pain. For restlessness hypodermic administra-

tion of sodium luminal, or administration of chloral hydrate by rectal instillation, is advised.

ADVANTAGES OF THE PROCEDURE.—(1) It allows direct approach to the abscess with some spillage or leakage of pus *on to* but not *into* brain substance, with avoidance of secondary suppurative encephalitis. With the wide open wound containing iodoform gauze kept wet either with Dakin or azochloramid solution, necrosis of the brain accompanied by suppuration does not occur. In fact, no actual pus is seen after complete evacuation of the abscess.

(2) Removal of the outer wall of the abscess allows complete inspection and cleansing of the abscess cavity so that a secondary pocket or an extension, should it exist, can be properly dealt with and drained.

(3) Cerebral and medullary compression are relieved by direct evacuation of the abscess, and recession of edema of the brain due to the open drainage avenue. Slow herniation, or progression of the floor of the abscess outward carrying the iodoform gauze with it, prevents compression during the time of rapid intraventricular accumulation of fluid.

(4) Should pericapsular brain necrosis coexist, it can easily be seen and the necrotic brain substance can be removed by suction.

(5) Meningitis does not occur (unless it already exists) by infection spreading to the meninges, for the reason that the meningeal spaces about the wound have been sealed off before the pus is evacuated.

(6) The disastrous results from ventricular perforation by a drainage tube, or dislodgment of the tube are avoided, because no tubes are used.

(7) When iodoform gauze is kept in contact with the inner surface of a non-encapsulated, or a weakly encapsulated abscess for a period of ten days, a definite protective fibrous layer develops on the surface or wall.

(8) Secondary pockets do not occur inasmuch as the remainder of the abscess is carried outward to, or about, the level of the skull, at which level it is maintained by lumbar puncture. The entire infected area is brought to the surface and is maintained at this level (Fig. 5). Multiple operations are obviated. The remaining portion of the capsule becomes a part of the scar tissue area at the operative site. Therefore, no scar tissue tract or closed sinus, with thick wall which might later act as a focus for recurrence, remains within the brain substance.

(9) Rupture of the ventricle from within outward, and overherniation are prevented by lumbar puncture. By inspection of the area under direct vision, one can determine the proper amount of fluid to be removed.

(10) The stuporous, semicomatose or comatose condition of the patient is relieved almost immediately and it does not recur. Therefore, early giving of fluids and food without having to resort to tubal feeding is carried out. This prevents choking and coughing which otherwise attend taking of food by a stuporous patient in the absence of tubal feeding.

(11) The procedure can be utilized in the majority of cerebral abscesses, and not only in small superficial abscesses as may be inferred from an illustration in my original paper.

These advantages are likewise obtained in the operation described by Horrax in those cases where it can be used. It can be used only in cases where there is a definite chronic abscess wall, and where the edges of the capsule can be brought out sufficiently to permit of suture to the galea.

CRITIQUE OF THE PROCEDURE.—(1) It cannot be utilized in cases of small, deep, and centrally placed abscesses. This is self-evident. One should be highly gratified if such an abscess could possibly be located, and still more so if it could be drained by any means. A small catheter introduced through the apparatus devised by Grant would be probably the method of choice.

(2) It should not be used in cases of cerebellar abscess except in those

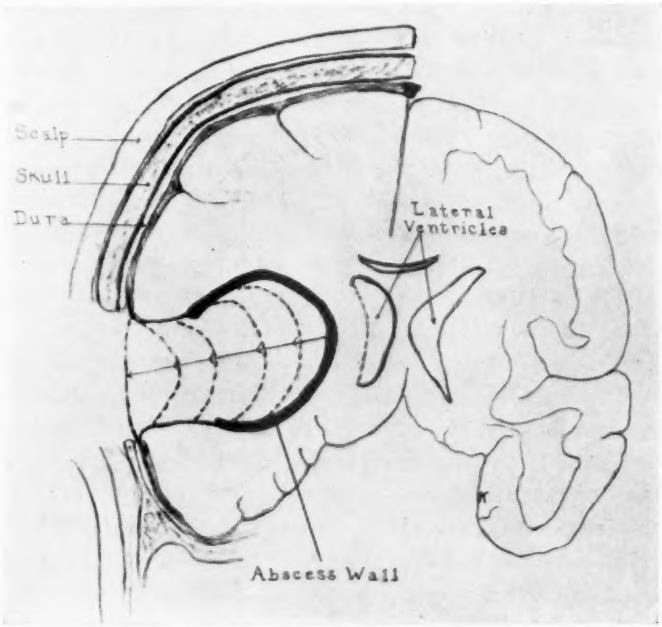


FIG. 5.—Illustration shows the gradual outward progress of the floor of the abscess cavity to the level of the skull, where it is maintained. One or more lumbar punctures may be necessary. The adjacent ventricle becomes markedly dilated, the opposite ventricle becomes moderately dilated. Shifting to the opposite side ceases and regression takes place.

in which the abscess is located completely within the cerebellar lobe, without being fixed to the petrus or dura.

(3) Objections have been made to the removal of a portion of the cortex overlying the abscess. There has been no instance of *production* of aphasia, mental deterioration, epileptiform attacks, or paralysis.

(4) Objection to the overherniation advocated in my original paper is proper. Not only was it unnecessary, but it required a longer period of time for eventual subsidence of the hernia and healing of the wound. Marked herniation is not allowed. When the floor of the cavity has reached the level of the skull, everything desired from herniation has been accomplished.

Frontal Lobe Abscess.—In the vast majority of cases abscesses of the frontal lobe follow infections of the nasal cavity and the accessory sinuses, with or without osteomyelitis of the frontal bone. They are usually ovoid in shape with the smaller end directed forward when the source of infection is the ethmoids or the frontal sinus. Most of them have but a small area of brain substance intervening between the anterior portion of the abscess wall and the inner table of the skull (posterior wall of the frontal sinus or ethmoids), and some may have perforated and drained into the nasal cavity. Those which develop secondary to osteomyelitis of the frontal bone are more likely to be spheroid. Eradication of the extracranial source of infection should be accomplished in all cases where the general condition of the patient will permit. Provided a definite diagnosis and localization of a frontal lobe abscess has been made, operation upon the frontal sinus and ethmoids, if necessary, should be done by the rhinologic surgeon and followed immediately by operation upon the abscess. If osteomyelitis of the frontal bone coexists, the complete operation for this condition according to a modified Furstenberg[16] procedure should be carried out. After removal of the involved frontal bone, an extradural pus pocket may be found. An opening through the dura leading into a subdural or subcortical abscess may be seen. If so, the abscess should be opened immediately. Otherwise, opening of an intact dura is better deferred unless one is certain that a subcortical abscess is present.

The incision begins in the outer extremity of the eyebrow, extends inward through the eyebrow to its inner extremity obliquely upward and inward to the midline of the forehead at the root of the nose, from which point it is carried vertically in the midline to within the hairline and thence downward for about two or two and one-half inches toward the temporal region. The flap consisting of all soft parts, except the pericranium, is turned down. The pericranium is included in the flap in cases of osteomyelitis of the frontal bone. This incision allows one to clean out the frontal sinus with preservation of the supra-orbital ridge, and to evacuate the frontal lobe abscess. Should it be necessary to exenterate the ethmoids, a short curved incision extending from the inner inferior angle of the incision is carried downward over the ethmoid region. The free end of the flap is turned underneath, thus folding the flap on itself, and is sutured loosely with the denuded surfaces approximated. This keeps the flap out of the way, controls bleeding from it, and prevents it from shriveling, as otherwise it would do if left free. After the floor of the abscess has herniated to the surface of the skull, it is maintained at this level by lumbar punctures, should they be necessary. When the surface has become firm and well granulated or covered with epithelium, the flap is unrolled and replaced in position. After healing, the only portion of the scar which can be seen is that in the midline. This is scarcely noticeable and may be altogether lost in the vertical wrinkles of the forehead. The other portions of the scar are within the eyebrow and the hairline and are not seen.

Should osteomyelitis of the frontal bone be present, the infected bone is removed according to a modified Furstenberg method through the same incision carried farther back of the hairline. Operation upon the frontal sinus and the ethmoids is done should it be necessary.

Most frontal lobe abscesses are fairly large, ovoid in shape, with the smaller pole directed forward and the larger backward. The wall of the abscess is thickest and toughest about its anterior pole where it approaches the cortex and dura. It is thinnest at the posterior pole which represents that portion of the abscess in its farther progress backward into the white substance of the brain. Should the abscess lie in the medial inferior portion of the frontal lobe, internal to the anterior horn of the lateral ventricle, it will collapse almost immediately after evacuation to such an extent that it is difficult to insert anything into the cavity without injuring its wall. In such instances, a fine meshed silver wire basket covered with a perforated rubber glove finger inserted into the cavity will serve to lead or guide the floor of the cavity to the surface and prevent collapse and subsequent pocketing. Herniation in this type of abscess will be marked if not controlled by lumbar puncture done after 48 hours. If the abscess is located in the outer upper portion of the frontal lobe external to and above the anterior horn of the lateral ventricle, it will not collapse readily. It is treated in the manner similar to that described for temporosphenoidal lobe abscess.

Although diagnosis of brain abscess is not discussed in this paper, the paralysis or paresis produced by frontal lobe abscess will be briefly considered. A large abscess situated in the outer upper portion of the lobe external to the anterior horn of the lateral sinus produces in its progress backward (if not operated upon) contralateral *paresis* of the upper extremity, then the lower extremity, and lastly the face. Such an abscess *per se* seldom produces paralysis for the reason that it is usually evacuated before this can occur, or it spontaneously ruptures into the ventricle. A sudden flaccid contralateral hemiplegia in which the patient's mental condition is not changed may be due to perforation of the posterior wall of the frontal sinus through the dura with extension of the infection upward and backward over the cortex along a narrow tract with formation of a well walled off subdural collection or pocket of pus over the motor area. In such an instance the spinal fluid cell count may be only six or eight. This paralysis clears up in about 48 hours after drainage of the walled off pocket of pus. In a case of suspected abscess, a sudden adverse change in the patient's mental condition either to stupor or coma, accompanied by high elevation of temperature and hemiplegia which develops rather rapidly and completely, is most likely due to rupture into the meninges with rapid spreading of pus over the cortex (massive suppurative leptomeningitis). A small superficial abscess situated in the anterior pole of the frontal lobe will more likely rupture and produce such a picture than a larger abscess which has progressed backward into the white substance of the brain.

CEREBELLAR ABSCESS.—A ventricular puncture should be made before an operation is performed for cerebellar abscess in order to lessen the intraventricular pressure, decrease the likelihood of jamming the conus into the foramen after evacuation of the abscess, and allow one to control the intraventricular pressure during convalescence. This puncture is advisedly made over the anterior horn of the right side for the reason that it gives good access for subsequent tappings of the ventricle and places the site of puncture away from the likelihood of infection.

In instances where a mastoid wound is not present, the approach to the abscess is made behind the lateral sinus. This allows clean exploration in case of negative findings. It also gives adequate exposure of the abscess if found. In the event that the lateral sinus has been opened for an infected thrombosis, or the abscess has perforated through the mastoid antrum or the lateral sinus, approach through the infected area would then be advisable. The procedure which I have described for cerebral abscess is advocated in the cases where the abscess is located centrally within the cerebellar lobe and is not fixed anteriorly to the petrus or dura. Such abscesses are more or less globular in shape and are in the minority.

Most cerebellar abscesses are located on the anterior inferior surface of the cerebellum and project into the lobe. They are usually quite ovoid in shape and resemble an elongated bird's egg. The smaller thickened pole of the capsule is attached to the dura or the petrus and is so fixed that no herniation can occur. In this type abscess the posterior pole of the wall or capsule of the abscess is exposed after the dura has been fixed to the cerebellum by sutures and electrocoagulation and removal of the overlying cortex by suction. A portion of the capsule about the size of a dime is removed with the electrosurgical needle, the cavity is washed out and inspected and two small rubber drainage tubes (Dakin tube size with a lateral perforation near the distal end) are inserted into the cavity to the full extent. The ends of the two tubes will completely fill the pointed distal extremity of the abscess cavity, while the remaining portion of the capsule will collapse about the tubes. No secondary pocket will develop. The opposite is the case when drainage tubes are placed in a frontal or temporosphenoidal lobe abscess cavity for the reason that the smaller pole or portion of the cavity is directed toward the operator and the larger pole of the abscess is toward the ventricle. The cavity may not collapse equally about the drainage tube, and a secondary pocket is likely to develop.

The incision in the scalp is closed at either extremity. The two tubes are brought out through the middle portion of the incision and about these the wound is loosely packed with iodoform gauze within the bony opening. The two tubes are fixed to the scalp to prevent displacement. In this type abscess there is no danger of perforation by the tubes. In subsequent dressings the tubes are irrigated with azochloramid solution, are allowed to remain in position for about three weeks, and then are gradually shortened until they are completely removed.

TRAUMATIC ABSCESS.—In cases of traumatic abscess, especially those secondary to compound, depressed fracture of the skull, the meninges have already become adherent and fused so that entry to the abscess cavity can be done without transgression of the meningeal spaces. Approach should be made through the scar tissue overlying the abscess where these spaces are obliterated without entering the free subdural or subarachnoid space. By so doing spreading of infection to these spaces and retraction of the cortex after evacuation of the abscess are not likely to occur. Should a foreign body be present within the cavity it is removed. Other foreign bodies lying without the abscess cavity in the brain had better be left unmolested at this time for fear of producing suppurative encephalitis. Should the abscess extend from the vertex downward to a depth below the level of the lateral ventricle, it will rapidly collapse after evacuation, so much so that it will be difficult to introduce any type of drain. Inasmuch as the cortex has already become firmly fixed to the dura, however, lumbar puncture may safely be done at the time of operation. As the cerebrospinal fluid is gradually removed through the lumbar puncture needle, one will observe that the abscess cavity enlarges again so that it can be thoroughly inspected. After this is done, iodoform gauze or plain gauze dipped in azochloramid oil is packed loosely into the cavity in the manner described. Intraventricular pressure is controlled by lumbar punctures, and the floor of the abscess cavity is allowed to progress slowly outward until it appears as a small dimple in the wound. This area granulates over and heals. In these cases the opening in the abscess may not be made as large as in the cerebral abscesses due to infections from the mastoid region or the nasal cavity and its accessory sinuses, for the reason that one desires to limit the size of the opening to the definitely sealed off region of the meninges. Although the duration of convalescence may be more prolonged, these cases can be gotten out of bed sooner than those with the usual type of abscess.

RESULTS

The rather numerous cases of fistulae and sinuses in the brain and traumatic abscesses developing about foreign bodies which were observed in military service in both Germany and the United States will be excluded.

Although a case was operated upon unintentionally in the manner described, in 1920, the procedure was not actually advocated until 1923. Nevertheless, all cases operated upon or seen by the author since 1920, with the exception of several cases of metastatic abscess, will be reported.

Thirty-two cases have been seen. Death resulted in the following groups of cases:

Three of the early cases in which a drainage tube was used; in one of which the tube perforated into the ventricle; in one the tube was dislodged and was replaced by an assistant into brain substance instead of into the tract; and in the third a secondary pocket ruptured into the ventricle.

One case in which a Mosher drain was used; cerebellar.

One case of acute abscess which was aspirated. The patient was moribund when seen; no drainage used.

Four cases of multiple abscess in which the second abscess was not located after operation upon one abscess, or in which a subcortical abscess was associated with a drained subdural pocket of pus.

One case of acute abscess associated with sinus thrombosis and blood stream infection.

Two cases operated upon for extensive suppurative leptomeningitis in which the presence or location of abscess was not determined.

One case of extensive suppurative leptomeningitis in which the presence and location of abscess was known before operation, but permission for operation at the time was not given. Moribund when explored.

One case of coexisting insufflation pneumonia following taking of food in a semicomatose condition. Moribund when explored.

One case in which the patient tore off the dressing and lacerated the hernia cerebri with her finger nails. Followed by infection of the brain.

One case, a child, of postoperative pneumonia.

Two cases without operation: one a few moments after being seen; cerebellar: the other 15 minutes before being seen due to spontaneous rupture of frontal lobe abscess into the ventricle.

Five of the fatal cases probably would have survived had proper management been carried out. The patient who removed the dressing was not under the operator's supervision, having been operated upon in a distant hospital. The child who developed pneumonia did so as a result of being dakinized and left by an open window. One child should have had a good chance of recovery had he been operated upon when the diagnosis was made, but permission was refused by both parents and physicians. The abscesses in two cases, associated with a subdural pocket of pus and a subcortical abscess respectively, which had previously been operated upon, probably could have been well localized and drained had ventriculograms been made.

Only three of the 18 cases enumerated above, in which a single abscess was present, were operated upon by the method advocated. All three were readily located, were well encapsulated, and were acceptably operated upon. One of these had insufflation pneumonia and was moribund when seen. Another is the patient who tore off her dressing, and the third is the child who died of pneumonia.

Three of the 17 cases in which the abscess was single, and who were operated upon by the method advocated, died, making a mortality rate of 17.5 per cent for this procedure. There was only one death in the last ten cases of this type consecutively operated upon.

Of the 14 cases which survived (Table I), the largest abscess contained more than seven ounces, and the smallest single abscess was about the size of a golf ball. The average size was about that of a lime or small tangerine. Five of the cases were either comatose or semicomatose at the time of opera-

tion. Four cases developed cerebrospinal leakage through the hernia cerebri, but with dakinization meningitis did not develop. None of these cases developed epilepsy following operation. One case, that of traumatic abscess in the parietal region, had epileptic attacks both before and following operation. No case had a permanent paresis or a hemi- or monoplegia following operation. Four cases operated upon by a colleague according to the principles set forth in this paper recovered; these cases are not included in this series.

TABLE I

RECOVERED CASES OF BRAIN ABSCESS

Case	Age	Sex	Source of Infection	Location	Pericapsular Brain Necrosis	Infection	Approximate Size
M. L.	28	M.	Mastoiditis with mastoidectomy	Left temporal lobe	None	Streptococcus	Tangerine
T. W.	46	M.	Infected C. C. F. of right frontal sinus	Right frontal lobe	None	*Streptococcus hemolyticus*	Duck egg
C. D.	26	M.	Mastoiditis with mastoidectomy	Left temporal lobe	Fairly well marked	*Staphylococcus aureus*	Tangerine
A. S.	22	M.	Infected C. C. F. of skull (gunshot)	Right parietal region	None	*Staphylococcus aureus*	Golf ball
G. F.	20	M.	Frontal sinusitis left	Right frontal lobe	None	*Staphylococcus aureus*	Large hen's egg
H. W.	19	F.	Mastoiditis with mastoidectomy and previous operation for brain abscess	Left temporal lobe	Mild	Not recorded (mixed infection from drainage tract)	Multiple (2) each size of malaga grape
C. B.	15	F.	Frontal sinusitis, bilateral; radical operation. Osteomyelitis of skull; operation	Right frontal lobe	Mild	Nonhemolytic streptococcus	Long-drawn-out hen's egg
P. M.	28	M.	Mastoiditis	Left temporal lobe	None	*Streptococcus mucosus capsulatus and B. coli communis*	Small orange
C. A.	6	F.	Mastoiditis	Left temporo-frontoparietal lobes	None	*Streptococcus hemolyticus*	Large orange, more than 7 ounces
L. N.	45	F.	Mastoiditis with mastoidectomy	Left temporal lobe	Marked	*Streptococcus hemolyticus*	Collapsed; spontaneous rupture into mastoid wound
L. B.	55	M.	Frontal sinusitis, ethmoiditis; radical frontal operation; old	Left frontal lobe	Marked	*Streptococcus hemolyticus*	Multiple (2) communicating. Small fig and butternut
F. R.	56	M.	Stab wound through old cranial defect	Left fronto-parietal lobes	None	Mixed. Discharging sinus	Elongated hen's egg
J. M.*	12	M.	Mastoiditis with mastoidectomy	Left cerebellar lobe	None	*Staphylococcus aureus*	Lime
L. A.	32	M.	Frontal sinusitis	Right frontal lobe	None	Specimen lost	Lime

* This patient was operated upon for his abscess by Dr. Daniel S. Cunning, who consulted the author regarding the patient's condition. The plastic procedure was performed by the author.

It is realized that this series of recovered cases is not large, but the fact that 14 out of 17 cases operated upon by this open method lived tends to make one who is accustomed to its use more confident in its efficacy. In fact, one can readily subscribe to the statement by Macewen that "One might almost conclude that in uncomplicated abscess of the brain, operated upon at a fairly early period, recovery ought to be the rule."

Until the probability or likelihood of brain abscess is suspected earlier than has been formerly the case so that diagnosis and localization can be made before a disastrous complication ensues, the mortality rate will continue to be fairly high, regardless of the type of operation used.

REFERENCES

[1] Adson, A. W., and Craig, W. McK.: Surgical Management of Brain Abscess. ANNALS OF SURGERY, vol. 101, pp. 7–26, 1935.

[2] Atkinson, E. Miles: Abscess of the Brain: Its Pathology, Diagnosis and Treatment. 289 pp. London, Medical Publications Ltd., 1934.

[3] Bagley, Jr., Charles: Brain Abscess with Pathological Observations. Surg., Gynec., and Obst., vol. 38, pp. 1–13, January, 1924.

[4] Cahill, H. P.: Twelve Cases of Cerebral and Cerebellar Abscess Drained by the Mosher Wire Gauze Cone. Tr. Am. Otol. Soc.

[5] Cahill, H. P.: Brain Abscess—Methods of Drainage. Tr. Am. Acad. Ophth. and Otol.

[6] Cahill, H. P.: Modern Treatment of Brain Abscess. J.A.M.A., vol. 102, pp. 273–276, January 27, 1934.

[7] Cahill, H. P.: Personal Communication to the Author.

[8] Coleman, C. C.: Some Observations on the Drainage of Subcortical Brain Abscess. Arch. Surg., vol. 10, pp. 212–216, January, 1925.

[9] Coleman, C. C.: Reduction of Mortality of Brain Abscess by Simple Methods of Treatment. South. Med. Jour., vol. 23, pp. 484–487, June, 1930.

[10] Dandy, W. E.: Treatment of Chronic Abscess of the Brain by Tapping: Preliminary Note. J.A.M.A., vol. 87, pp. 1477–1478, October 30, 1926.

[11] Dandy, W. E.: Lewis' Practice of Surgery, vol. 12. W. F. Prior Co., Inc., Hagerstown, 1934.

[12] Davidoff, L. M.: How to Obviate Failures in the Results of Surgery of Brain Abscess. Laryngoscope, vol. 44, pp. 871–875, November, 1934.

[13] Dowman, C. E.: The Treatment of Brain Abscess by the Induction of Protective Adhesions between the Brain Cortex and the Dura before the Establishment of Drainage. Arch. Surg., vol. 6, pp. 747–754, May, 1923.

[14] Eagleton, W. P.: Brain Abscess: Its Surgical Pathology and Operative Technic. 297 pp., The Macmillan Co., New York, 1922.

[15] Evans, William: The Pathology and Aetiology of Brain Abscess. Lancet, vol. 1, pp. 1231–1235, June 6; pp. 1289–1293, June 13, 1931.

[16] Furstenberg, A. C.: Osteomyelitis of the Skull: Osteogenetic Processes in the Repair of Cranial Defects. Ann. Otol., Rhinol., and Laryngol., vol. 40, pp. 996–1012, December, 1931.

[17] Grant, F. C.: The Mortality from Abscess of the Brain. J.A.M.A., vol. 99, pp. 550–556, August 13, 1932.

[18] Horrax, Gilbert: A Method for the Treatment of Certain Chronic Encapsulated Brain Abscesses. Surg. Clin. N. Amer., pp. 1179–1186, October, 1934.

[19] King, J. E. J.: The Treatment of Brain Abscess by Unroofing and Temporary Herniation of Abscess Cavity with the Avoidance of Usual Drainage Methods. Surg., Gynec., and Obst., vol. 39, pp. 554–568, November, 1924.

[20] King, J. E. J.: Brain Abscess—External Rupture of "Capsule" with Pericapsular Brain Necrosis. ANNALS OF SURGERY, vol. 101, pp. 190–200, January, 1935.

[21] Macewen, William: Pyogenic Infective Diseases of the Brain and Spinal Cord. 354 pp., The Macmillan Co., New York, 1893.

[22] McKenzie, K. G.: Treatment of Abscess of the Brain. Arch. Surg., vol. 18, pp. 1594–1620, April, 1929.

[23] Penfield, W.: Personal Communication quoted by Adson.

[24] Sachs, Ernest: The Diagnosis and Treatment of Brain Tumors. 396 pp. The C. V. Mosby Co., St. Louis, 1931.

DISCUSSION.—DR. CLAUDE C. COLEMAN (Richmond, Va.).—There has been some unanimity of opinion regarding the treatment of brain abscess. It is generally believed that abscess of the brain does not become operative until encapsulation takes place. In the first stage of abscess formation the condition is a diffuse, septic encephalitis which cannot be drained, and, therefore, attempts at drainage have proved to be harmful, as no effective surgical measures have been devised for the eradication of the infection. One must wait, therefore, for encapsulation before attempting to eradicate the abscess.

While brain abscess is fatal in its tendencies if not operated upon, and while a large majority of patients will die unless the abscess is eradicated, the fact remains that when a patient does die following operation, he generally dies because of the surgeon's well intended efforts to remove the abscess. In other words, fatalities following brain abscess operations are usually the result of spreading the infection into the unprotected brain tissues outside of the capsule.

For a number of years I have used one of the methods to which Doctor King referred. That is, drainage with the eye end of a soft rubber catheter. If there is much difficulty in placing the catheter into the abscess cavity, the fumbling with the catheter is likely to lead to an extravasation of pus outside the capsule and a diffuse encephalitis, with secondary abscess formation. We have been more concerned with and troubled by the small, deeply seated, densely encapsulated brain abscess than any other type of encapsulated abscess. In some cases, particularly deeply seated abscesses, in which the capsule is dense, we have first verified the presence and location of the abscess by means of a ventricular needle over which a cannula is fitted. If the capsule of a deeply seated abscess offers much resistance to the penetration of the ventricular needle, we have slipped the silver cannula into the abscess cavity as soon as the pus began to escape from the needle, and left the cannula for permanent drainage instead of using a catheter. In two of the cases treated by drainage with a soft rubber catheter, the abscess cavity, as determined by the depth of penetration of the catheter, appeared to extend across the median line. This was due to the swelling of the brain on the side of the abscess, with displacement of the falx to the opposite side.

I should like to emphasize what I believe to be a logical attitude toward the treatment of brain abscess; that there is no standardized treatment which will be applicable to all cases. In superficial abscesses with a thin layer of brain tissue between the dura and abscess cavity, I should expect excellent results from Doctor King's "unroofing" operation. In deeply seated densely encapsulated abscesses, one must rely upon simple tapping, as Dandy has advocated, or upon some method of drainage that will empty the abscess with the least disturbance of the surrounding brain. The method of drainage with the eye end of a small, soft rubber catheter or with a cannula fitted over a ventricular needle, for the deeply seated abscess (and many of them are of

this kind), has given about as satisfactory results as any method we have seen reported.

Dr. Joseph E. J. King (closing).—As I have stated before, 14 cases out of 17 which were operated upon according to the method described survived, and we have seen an autopsy performed upon only one of these 14. An old man, who was the second in the series of those who survived, and who was an habitual alcoholic, was admitted to Bellevue Hospital three or four years later in an alcoholic stupor, and died. The autopsy revealed no pathologic condition in his brain other than a dilated anterior horn of the right side beneath the site of the operation for a frontal lobe abscess.

None of the cases had epilepsy other than the fourth case who had a traumatic abscess in the right parietal region and who had epileptic attacks before operation.

I agree with Doctor Coleman that if operation can be delayed until a capsule is formed, it is safer than operating in the stage of suppurative encephalitis. In one case of metastatic abscess, which is not included in this series, we were advised by the attending neurologist to operate upon him early, and the patient was fortunate enough to survive. In a previous paper by Doctor Coleman he stated that four out of five deaths were due to leakage about the drainage tube with formation of suppurative encephalitis, which condition resulted in death.

I stated that one would be fortunate to locate a very deep seated abscess and to drain it by any means, but preferably by the small catheter as described by Coleman, or a searcher or cannula, and one would be most fortunate to be able to drain it by any method. I have never advocated the procedure described in this paper for a very deep seated small abscess. The procedure described is intended for abscesses which extend to within 3 or 4 cm. of the surface, exclusive of the basilar surface. Abscesses of this type offer the best opportunity for a successful outcome.

THE SURGICAL TREATMENT OF EPILEPTIFORM SEIZURES

LOYAL DAVIS, M.D., AND WILLIAM DROEGEMUELLER, M.D.

CHICAGO, ILL.

FROM THE DIVISION OF SURGERY, NORTHWESTERN UNIVERSITY MEDICAL SCHOOL, CHICAGO, ILL.

THOUGH the diagnosis of epilepsy in most of its multiple and varied manifestations is easy, its underlying nervous physiology is obscure and difficult to comprehend. That epilepsy is not a disease is agreed upon. The clinical entity is the result of a sudden, violent discharge of neural elements produced by a noxious stimulus. As Hughlings Jackson expressed it, "an experiment made upon the brain by disease."

There is a form of epilepsy, by some termed "idiopathic" or "true" epilepsy, which has as its only symptom convulsive seizures. No anatomic substratum has ever been discovered for this type of epilepsy. Then there are diseases which have convulsive seizures as a part, and often only a small part, of their symptomatology; for example, intracranial tumors, abscesses, arachnoiditis, cerebral arteriosclerosis and intoxications. Such epileptiform seizures must be differentiated from true epilepsy of unknown etiology. It is with the pathologic conditions accompanied by epileptiform or convulsive seizures that surgical therapy is concerned.

An epileptiform seizure may include symptoms the result of activity of the cortical motor area; the so called sensory area; or the cerebral visual, auditory, olfactory, gustatory, and autonomic centers. In accepting such a conception, there is no difference between a localized motor spasm, paresthesia of a limb, hallucinations of sound, smell or sight and certain visceral and autonomic phenomena. All are epileptiform seizures, which may usher in a complete convulsive attack, and under such circumstances may be regarded as an aura.

The continued search for the physiologic facts of epilepsy has led to acceptance of the basic unity of the convulsive state as well as to the astonishing variety of stimuli which may produce such episodes. Though the symptoms be protean in character and the nature of the stimulus varied, it is probable that there is a common, connecting neural mechanism.

Since the discovery by Fritsch and Hitzig of the electrical irritability of the brain, numerous theories concerning the site of convulsive discharge have been proposed. It is unfortunate for the clarity of our knowledge that this site has been considered inseparable from the pathogenesis of the seizures. It is further unfortunate that much of the work has been concerned with the establishment of a so called convulsive center.

Three chief theories have been proposed concerning the location of a convulsive site. First, that which deals with the cortex alone; second, that which includes the cortex and subcortex; and third, the medullary theory. The experiments performed to prove any of these theories have been of two

types; first, those in which convulsive seizures have been produced by irritation of various parts of the central nervous system, and second, those in which experimentally produced convulsions have been modified, stopped or prevented by ablation of certain parts of the nervous system.

The proponents of the medullary theory have assumed that if convulsions may be produced by irritation of the pons and medulla, and may occur as the result of intoxication with absinthe in decerebrated animals, the convulsive site either exists in the pons and medulla, or the pons and medulla possess epileptogenous properties. Opposed to this theory are the observations that when a convulsion occurs as the result of irritation of the pons, the clonic element disappears after removal of the cerebral cortex; and that removal of one cerebral hemisphere causes convulsions to disappear from one-half of the body.

Those who support the mixed theory are dependent upon observations of certain motor manifestations produced by stimulation of various parts of the central nervous system and many of these experiments have nothing to do with convulsions. They are concerned with the production of certain forced movements, ataxia and dystonia.

The evidence which has supported the theory of cortical origin has been supplied chiefly by the observation of convulsions produced by electrical stimulation of the cortex. It has been found that convulsions may be produced which resemble epilepsy. It has also been shown that if electrical stimulation of the motor cortex is long continued, the resulting isolated contractions of muscle groups become successive and spread to muscle groups innervated by adjacent cortical areas; then spread to the other side with the resulting convulsion outlasting the electrical stimulation. If an irritated zone be extirpated quickly, the convulsion is inhibited. After deep decortication, such convulsions cannot be produced, and when certain motor areas have been removed the movements represented by such an area are absent in an ensuing convulsion. Not only can such convulsions be produced by irritation of the motor cortex, but by stimulation of other cortical areas such as the occipital lobe.

From our own studies,[1] and from the accumulated reports of experimental work, it may be stated that whatever element of a seizure may be lacking in a convulsion produced in an animal with a mutilated brain, convulsions (tonic, clonic, or both) may be produced from the cortex and subcortical region and, in the latter case, in the absence of the motor cortex. The symptoms of a convulsion vary with the state of preservation of the brain. If it be intact, tonic-clonic convulsions associated with sialorrhea, pupillary changes, bladder contraction, circulatory and respiratory changes, and at times an aura may be present. The aura is dependent upon the area of brain irritated; and in jacksonian attacks, as in artificially produced cortical seizures, motor movements constitute the aura. In other cases, a vagal aura exists, respiratory or circulatory changes precede the convulsion, and the portion of the brain irritated must be that of a lower level. It may be pointed out that

not only is the tonic element of an epileptiform seizure absent in a cortical fit, but loss of consciousness does not occur until a generalized convulsion ensues, and involuntary urination does not occur before the loss of consciousness. A major epileptic convulsion consists not only of tonic and clonic movements, but also of a series of events including the aura, vascular, respiratory, secretory changes, and contraction of the involuntary muscles of the bladder.

The earliest proven adventure of man into the field of major surgery was trepanation for fits, and so it may be said that man's experience with the surgical treatment of the convulsive state began at least a quarter of a million years ago. Though the progress of surgical therapy waxed and waned, the problem of epilepsy has remained persistently for solution. The contribution of Hippocrates to the treatment of injuries of the head gave rise to a literature dealing with the surgical aspects of traumatic convulsions which grew to an enormous proportion by the time of the sixteenth century. To Guy de Chauliac epilepsy was simply a convulsion of the entire body arising from excess of humidity and "it suffices the surgeon to know what has been said of them, their symptoms and cure, if there is any." The character of these surgical procedures may be judged fairly well when it is realized that even during the seventeenth century Thomas Willis struggled valiantly and alone to wean his more philosophic colleagues from an unshaken belief in the rôle of Satan in convulsive states by constantly centering their attention upon the central nervous system.

Our own experience with surgical procedures in convulsive states has been obtained from two types of patients. First, those who have had convulsions produced by intracranial tumors or abscesses, and second, those whose seizures were supposedly the result of trauma to the brain.

In reviewing our material, only cases verified pathologically at operation or by autopsy examination were included. In all but a very few of the cases the pathologic findings rest upon the observations made at operation; in some instances these are supported by an autopsy examination. The remaining cases were studied, though an operation had not been performed, because of a complete description of the pathology found at autopsy. The review of our case records has made it apparent that the material is not so detailed as it might have been had the patients been studied originally with the idea in mind of making a report upon their epileptiform manifestations. For example, we do not have the evidence available nor are we fitted by training or experience to include very important data concerning the so called "epileptic constitution" or "make-up" of our patients. Why do convulsions occur in one patient with an intracranial tumor and not in another patient with a tumor similar in type and location? Similarly, does not the "potentially an epileptic" factor play an important rôle in the development of convulsions in the approximately five patients out of a hundred, all of whom have received severe wounds of the brain?

Obviously, it is not entirely satisfactory to classify the location of the

pathologic changes on the basis of what is observed at operation. Adjacent portions of the brain may be involved in the absence of gross changes in appearance. With proper emphasis upon this reservation, the operative records and sketches, with the exception of those cases in which an autopsy was performed, constitute the basis for localization.

We included in the study of our material all patients with symptoms which to us represented epileptiform manifestations or a convulsive state. Periods of sudden unconsciousness, not syncope; periods of twitching, or drawing of the hand or foot; peculiar odors with periods of apprehension are as significant to us as major seizures; in fact, many of these minor spells are of more localizing and diagnostic value. Many papers in the literature reporting upon the occurrence of convulsions in groups of intracranial neoplasms exclude this important group.

It is exceedingly difficult to obtain an accurate description of a seizure. To describe all its manifestations, if one has the opportunity to observe an attack, is often impossible. Patients are often told what went on after they lost consciousness and many important details of the symptoms are lost. Patients or relatives may regard or neglect minor manifestations as insignificant in the presence of more dramatic symptoms.

Rarely did the seizures occur in a definite period of frequency. However, in spite of the irregularity of occurrence, for comparison they could be grouped roughly into weekly, monthly, or infrequent and single episodes. Postoperatively, the same grouping as to the frequency of attacks was made.

It is in this respect that it is often difficult to compare one case directly with another. It is of more value to compare the condition of the patient before and after operation with and without medical treatment. Is the patient as well off after operation as he was before operation? Can it be said in a given case that the surgical procedure made it impossible to control the convulsions more successfully by adequate bromide or luminal therapy?*

It is somewhat of a surprise to realize the large number of patients with intracranial tumors who have convulsions, and particularly the large number in whom a convulsion was the initial symptom which first called the attention of the patient or his family to his difficulty. It was not long ago that the general impression existed, and it was taught, that the meningiomata were the intracranial tumors most often associated with convulsions. In our own experience, 62 per cent of the patients with glioblastomata; 72 per cent of the patients with astrocytomata; 39 per cent of the patients with meningiomata, and all but one patient of the angioma group presented epileptiform seizures. Four patients of 36 who had pituitary tumors had convulsions, but it should be noted that in each of these patients the tumor had grown outside of the boundaries of the sella turcica. Thirty-seven per cent of our

* By adequate bromide therapy we mean the administration of bromides under careful control of the bromine content of the blood. The variation of the bromine level in the blood necessary to control convulsions in patients has been well established.

patients with metastatic intracranial tumors presented attacks of one type or another.

Neither can the meningiomata be said to be the only group of intracranial tumors which are accompanied by a history of convulsions of long standing. Whereas 58.5 per cent of our meningiomata gave a history of seizures lasting from one to six years or more, likewise the glioblastomata (35 per cent), astrocytomata (57 per cent), and angiomata (75 per cent) presented a history of attacks over an equally long period.

In any series of intracranial tumors, the majority of patients are adults. It is important, therefore, to point out and to reemphasize the fact that convulsions occurring in an adult should be immediately suspected of being due to an intracranial tumor. It remains, therefore, a duty of the physician to bear this in mind and to use every possible diagnostic aid at his command to prove that his patient does not have a space-occupying lesion.

Our records show that although 39 per cent of the meningiomata presented convulsions, choked disks (that symptom which many believe must be present before a diagnosis of an intracranial tumor can be made) were absent in 36 per cent of the patients. Likewise, choked disks were absent in 28 per cent of the astrocytomata; in 17 per cent of the glioblastomata, and in all of the angiomata. Again it should be emphasized that choked disks are a late symptom in a large number of intracranial tumors. We do not mean to state that every adult who has convulsions has an intracranial tumor, but it cannot be repeated too often that to prove that the patient does not have an intracranial tumor is the duty of the physician and not the individual.

Intracranial abscesses may be considered in the same group as the intracranial tumors in relation to the occurrence of epileptic manifestations. Of our 42 patients in the abscess group, 15 had convulsions. Of these 15, five are still living and three of these five still have convulsive seizures which must be controlled by bromide therapy. Twelve of the 42 patients are still living and none of these 12 had seizures after operation who did not have them in the acute stage of their illness before operation. The period of time during which seizures were present before operation was very definitely shorter than that found in the intracranial tumor cases and this can easily be explained by the more acute nature of the pathology present.

Our material was studied to determine whether or not any relation existed between the occurrence of epileptiform seizures and the location of the tumor or abscess. Quite in keeping with what our general ideas have been from our clinical experiences and experimental investigations, we found that seizures occurred in patients with tumors which were situated in the cortex, subcortex, pons, midbrain, third ventricle, and posterior fossa. The only tumors which occur in the posterior fossa and which were not accompanied by convulsive seizures in the patients in our series were the acoustic neurinomata.

An attempt was also made to relate certain epileptic manifestations with definite functional areas in the brain. It should be immediately recognized

that this is just as difficult as it is to draw conclusions concerning cerebral localization from tumor or abscess pathologic material. Both at operation and autopsy it is hard, particularly in the gliomata, to determine the limits of the tumor growth. Although a meningioma does not involve the brain directly, the extent of damage produced by pressure is equally as difficult to determine. So, we have been able only to make certain rather general deductions in relation to gross divisions of the brain. After analyzing all of the cases for objective and subjective phenomena presented in the described seizures, a very few facts stand out. For example, as compared to the frontal, temporal and the occipital lobes, localized muscular twitchings, commonly described as a jacksonian attack, occurred by far more often in these tumors situated in the parietal lobe. On the contrary, when such objective symptoms as pallor, drooling, cyanosis, flushing, or lacrimation occur, all of which might be grouped as vasomotor phenomena, they were found to be present in tumors in the frontal or temporal lobes and not in tumors of the parietal or occipital lobes. Loss of consciousness occurred just as often in the tumors of one lobe as of another. We cannot subscribe completely, therefore, to the view that arrest of consciousness occurs when the frontal lobe alone is discharging.

Likewise, it is obvious from our material that the subjective epileptic manifestation of a bad odor or taste occurred exclusively in tumors of the temporal lobe and, more particularly, in tumors which involved the medial aspect of that lobe, the uncinate gyrus. The subjective symptom crudely described by the patient as "tingling, numbness, or electric shock" occurred more often in parietal lobe tumors situated within or posterior to the postcentral convolution; but in two instances of frontal lobe and in the same number of temporal lobe tumors, the patient described a similar sensation preceding his attack. Here, of course, lies the danger of stating that this symptom occurs in temporal or frontal lobe tumors, because only by careful pathologic examination of the entire brain could one be justified in stating that the postcentral convolution of the parietal lobe was not involved by the tumor. Such a circumstance could also be explained, of course, by an involvement of one of the large cerebral arteries so that an epileptiform seizure might begin in any portion of the brain at a distance from the neoplasm but in the area of distribution of the artery. We have found it to be true in every instance that the patient who had a visual aura had a tumor in the occipital lobe. The exception to this has been those patients with tumors in the temporal lobe whose aura was one involving objects rather than light or color. We have seen temporary complete blindness during the discharge of the occipital lobe produced by a tumor, and this has been demonstrated by others.

Penfield[2] has reported the occurrence of "autonomic epilepsy" which occurred in a patient with a tumor of the third ventricle. It would seem from this case that a convulsive discharge may occur from centers as high as the anterior portions of the thalamus. The reports of other patients with sensory

epileptiform seizures in which the tingling spread gradually through one side of the body and became so severe as to be painful also suggests that the gray matter of the thalamus may become involved even if the original discharge does not take place there.

It has been our experience that immediately preceding a convulsive seizure, deviation of the head and eyes occurs toward the side of the lesion, that is, toward the site of irritation. This is not in accord with statements of some individuals who feel that the most frequent lateralizing sign is deviation of the head and eyes to the side opposite the hemisphere involved. It is important to attempt to localize epileptic manifestations within the brain so that convulsive seizures can be accurately interpreted, as an aid both to diagnosis and therapeusis, but we doubt very much whether or not definite epileptiform manifestations can be ascribed solely and specifically to certain lobes or gyri of the brain. However, we are able to confirm the clinical fact which has been known for some time that the epileptic manifestations of a tumor in the posterior fossa are very definite. They have been described previously as "cerebellar fits." The outstanding characteristic of these convulsive states is the occurrence of a tonic convulsion involving all four extremities, opisthotonos, with marked respiratory and vasomotor symptoms. Convulsions have occurred in eight patients with posterior fossa tumors. Three of these were children who had medulloblastomata.

When one begins to determine the effect of removal of an intracranial tumor upon the occurrence of convulsive attacks, several factors arise which interfere with the collection of data of accurate scientific value. It is not always possible to determine the frequency or the nature of seizures in a given patient before operation under the conditions of accurately controlled bromide or luminal therapy, or without therapy. Likewise, it is not always possible after operation to allow a patient to go for a period untreated with medicine in order to make a comparison with his condition under treatment. It is very seldom that the patient with epileptiform seizures is observed over a sufficiently long period before operation by the surgeon who removes his intracranial tumor. This introduces the factor of the judgment of several clinicians as to severity, frequency, and nature of the attacks before and after operation.

We have attempted to study all of our intracranial tumor patients in an effort to determine whether the attacks were more frequent after operation; whether they remained the same; whether they were less in frequency; or whether they did not recur. As was stated, these figures can be taken only in a general way and represent patients who were not given bromide or luminal therapy after operation. Of the patients with glioblastomata, and it must be remembered that the life expectancy of patients with this type of tumor after operation is on the average of 6 to 18 months, 10 per cent of those having attacks prior to operation had none after operation up until the time of death, and 32 per cent had fewer and less severe attacks following operation. Of the astrocytoma group, 31 per cent were judged to be unim-

proved as far as their convulsions were concerned. Fifty per cent had fewer and less severe attacks and 6 per cent had no attacks following operation over an interval of 3 and 6 years, respectively. These figures represent about the condition which exists in the other tumors in the glioma group.

Of the meningioma group, and in these patients the tumor was removed completely, three patients were definitely worse following operation and finally had to be placed on bromide or luminal therapy. Eight patients had fewer attacks which were less severe, but were not entirely free, in spite of the fact that their tumors had been completely removed. The remaining five of the 16 patients in the group who had convulsions before operation had no further convulsions following removal of their tumor and have been without bromide or luminal therapy. It becomes apparent that although these tumors have been removed completely certain changes must have been produced within the brain which continue to act as irritative foci which release a convulsive discharge. These changes may be and probably are microscopic in nature and further surgery under such circumstances is illogical and contraindicated. Particularly is this true when these attacks can be so well controlled by judicious medicinal therapy.

In general, one may say without a doubt that the removal of an intracranial tumor is followed in the majority of patients by definite improvement in the frequency and severity of the convulsive seizures. It cannot be guaranteed, however, that the attacks will completely disappear. It is now our custom to begin bromide or luminal therapy preoperatively upon those patients who give a history of convulsive attacks and to continue that therapy after operation. By maintaining a constant bromine level in the blood the patient's attacks can be stopped and gradually the dosage of bromides be reduced.

As our records show, successful drainage of a brain abscess may be followed in later years by the development of epileptic seizures and the pathologic changes which occur in the brain during the obliteration of the abscess cavity provide a focus from which the convulsive discharge may occur.

A second group of patients with convulsive seizures are those in whom trauma to the brain has played an important rôle. Here again, before the exact rôle of trauma can be evaluated in each case a study should be made to determine what part the constitutional makeup of the individual has played in the development of the epilepsy. This is particularly emphasized by the statistics which have been gathered by a number of men relating to the occurrence of epileptiform attacks following trauma to the brain. In a group of 18,000 cases of gunshot wounds of the head, Turner finds that less than 5 per cent of the patients developed seizures. The statistics vary above and below that figure and, of course, it must be realized that the cases studied are not directly comparable. The material, no less than the observers, varies so greatly that it is difficult to find a common basis for reckoning the percentage of instances. However, 5 per cent may be taken as a reasonable percentage of the occurrence of convulsions after trauma to the brain.

Granted that the character and severity of trauma in the group of cases is comparable, how is one to explain the fact that the remaining 95 per cent of patients never develop epileptic seizures?

It has been well established that when the brain is injured and the damaged cerebral tissue is not removed, a scar of fibrous astrocytes, connective tissue and newly formed vessels develops. This scar soon becomes densely adherent to the overlying meninges. The rich plexus of blood vessels anastomose with the cerebral vessels on one side and with the vessels of the meninges and scalp on the other. Like all cicatricial tissue, this scar contracts and a pull upon the framework of glial and vascular tissue develops. This cicatricial pull is not confined to the local area of scar tissue; the homolateral hemisphere and even the entire brain is affected.

Foerster[3] has described the mechanism of the pull of such a cicatrix. He noted a displacement of both lateral and third ventricles toward the site of the scar tissue with enlargement of the lateral ventricle upon the side of the lesion. There is a certain amount of cerebral atrophy which accompanies the formation of scar tissue, but it probably plays no important rôle in the ventricular displacement.

Penfield's[4] examination of the scar tissue removed invariably showed fibrous tissue near the surface of the cortex and meningeal adhesions. The fibers of astrocytes were arranged parallel to the direction of traction and extended upward toward the scar. Deep within the brain the astrocytes and blood vessels formed the only framework capable of resisting the constant pull of the contracting cicatrix. In cases of long standing, tubes and shoots of neuroglia surrounded by connective tissue sheaths may result from the progressive cicatrization. We have seen gelatinous, yellowish-gray fibrous tissue as the center of an area of atrophy and cyst formation in old cases; a fact which Foerster and Penfield[4] record, emphasizing the focus of concentric traction.

The pathogenic relation between the formation of the scar, the development of a vascular plexus, the steady pull of the contracting scar and epileptic attacks are immediately suggested. Foerster and Penfield have expressed the problem very clearly: "If the stimulus for an epileptic seizure arises in the nervous elements one might expect the attack to begin shortly after the infliction of the wound when these elements are plentiful and undergoing progressive destruction . . . If, on the other hand, the vascular elements are responsible for the initiation of the process, epilepsy might well be expected to make its appearance later, when the vascular plexus reaches its fullest development and nerve fibers have largely vanished.

"It has been shown that at operation the focal epileptic attacks may often be produced in two ways: either by electrical stimulation of the brain in the neighborhood of the wound, or by gently pulling upon the adherent dura. This latter fact may be of considerable significance, for, if the increase of a pre-existing strain produces an attack, it may well be that the

pre-existing strain itself is an important factor in the etiology of spontaneous convulsions.

"As pointed out above, the blood-vessels form in one sense the roof of the contracting network. Traction, therefore, upon the vessels must be inevitable. The hypothesis at once suggests itself that a vasomotor reflex secondary to this traction is responsible for the initiation of the convulsive seizures."

The radical excision of one of these extensive, contracting scars for the relief of convulsions, assuming that the mechanism just described constitutes the pathogenesis of the attacks, can be a rational procedure only if the damage produced by excision is not accompanied by the same train of histologic events that followed the original lesion. Penfield's[5] histologic studies made upon the excision of cerebral tissue would point to an entirely different result. He has stated that when brain tissue is excised, there is no organization of connective tissue. Instead, a fluid-filled space with a small amount of gliosis about it appears. There is no plexus of newly formed vessels and there is no evidence of a cicatricial pull upon the surrounding brain tissue. Brain injury and brain excision are followed by entirely different structural changes.

If this were the entire story underlying the surgical relief of epileptiform seizures, the problem would be relatively simple. Only the careful study of patients, as human experimental problems, will answer the question of the wisdom of substituting a non-contracting fluid-filled space for a contracting cicatrix. One must assume, in such a working hypothesis, that removal of an apparently gross area of irritation will cause the entire neural mechanism responsible for a convulsion to disappear. This question has not been answered as yet by clinical cases carefully studied over a sufficiently long period. However, a valuable suggestion for the prophylactic treatment of severe brain and skull injuries has been made. The careful painstaking débridement of compounded craniocerebral injuries might reasonably be expected to decrease the incidence of posttraumatic convulsive attacks.

Our experiences with posttraumatic seizures have been purposely limited to those individuals who have organic evidences of the severity of their original injury; those who have organic symptoms following their convulsions and those in whom encephalographic studies demonstrate a mechanical defect which surgical intervention may be expected to remedy. This means that we have never attempted to localize the site of a suspected focus by an interpretation of the pattern of the attack alone, and then subsequently excise that area of cortical tissue. The physiologic interpretation of our own experiments on convulsions as well as our clinical observations lead us to believe that in the present state of our knowledge such a procedure is unjustified.

The material includes 13 patients whose attacks were attributed to craniocerebral compounded wounds due to blows on the head by bricks, pipes, hammers; bullet wounds, or automobile injuries; four patients with perencephalic cavities some of which were associated with a congenital skull defect;

one patient with a definite history of birth injury; and five patients with convulsions which followed encephalitis, all of whom had focal attacks with organic symptoms of the lesion.

In contrast to the 13 patients with severe compounded craniocerebral wounds with convulsions, we have observed and treated 32 other patients all of whom had gross cerebral damage, and none of whom has as yet developed convulsive seizures. However, this time limit apparently never runs out. Six of the 13 patients had an interval of over six years between the injury and their first attack. The longest interval was 21 years and the shortest six months.

Apparently the site of injury and the length of the period of coma have no bearing upon the subsequent course of events. Five of our severely injured patients had no loss of consciousness; three were in coma a few hours and five were unconscious for from 12 hours to a month. The site of injury was equally distributed between the frontal, parietal, temporal, and occipital areas of the skull.

The convulsive attacks in this group of 13 patients were predominantly major in character in eight instances. That is to say, the attacks began as focal seizures, then became generalized and were accompanied by loss of consciousness. In the remaining five patients, the attacks were characterized by momentary dreamy states or localized muscular twitchings. The frequency of attacks varied from several each day to one every six months. Two patients had repeated attacks and then without any apparent reason had intervals of as long as six and ten years without an attack, only to develop a status epilepticus.

At operation several varieties of pathologic changes were observed in this group of cases. In all of those in which there had been gross damage to the scalp, skull and brain, dense adhesions were found between the dura mater, brain and skull. In several instances the skull defect had caused the underlying brain to become firmly attached to the scalp. As the scalp flap was elevated, the scar tissue pull would bring the brain along with it. We found all grades of arachnoidal thickening, often so opaque that the cerebrospinal fluid accumulated in lakes along the sulci between the convolutions. Often the fluid so trapped was yellow in color, further evidence of the lack of circulation in the subarachnoid space.

Increased vascularity of the dura mater and cicatrized brain tissue was a constant finding. In one patient, who had received a stellate-shaped depressed fracture of the temporal bone 21 years before her first attack, large arteries and veins formed suspension cables in some areas between the cortex and dura mater. Associated with these more or less external changes, we invariably have found an area of cortical cicatrization, thick, firm, grayish-yellow in color and easily recognizable in differentiation from the normal brain tissue. Often the center of this cone shaped tissue was soft, gelatinous and cystic.

Such was the condition found in a patient who received a severe skull

injury as a boy and who had his first convulsive seizure eight years later. After an interval of another 13 years during which convulsions occurred at irregular intervals, he developed weakness in the right face, arm and leg and presented himself for treatment. At operation the cortical scar was blocked out and removed en masse. In spite of a period of freedom from attacks for eight months, the convulsions returned. He was placed on carefully controlled therapy but died in status epilepticus. The appearance of the brain after surgical removal of the originally damaged tissue approximates very closely the condition found at operation except for the smaller size of the scar.

In two cases we removed a foreign body in the center of the fibrotic tissue; in one instance a screw and in another a bullet, with complete relief of the seizures. In two patients, rib transplantation was performed to prevent recurrence of the adhesion between the scalp and dura mater. One of these latter patients is now free from convulsions though it is necessary for him to take 30 grains of bromides daily in order to keep his blood bromine content at 125 mg., which for him is the nonconvulsant level. In one instance we removed a large calcified mass from within the cortex which had evidently developed from an area of hemorrhage, and the patient has had no more attacks though he has never received bromides.

In evaluating the results following operation we find that there was no improvement in one patient; another died in status epilepticus after an eight months' period of freedom; another patient has had no attacks in nine years, though it must be noted that there was a free interval of six years before operation. In one patient the character of the attacks has changed. For six years he has had no major convulsive attacks but "dreamy states" occur about once every six to eight weeks. Other patients have been free from attacks for a year or less without medication.

Our results in those patients with large cortical defects, following injury at birth or accompanied by congenital defects in the skull, have been equivocal. The most satisfactory experience occurred in a boy of ten who had a left hemiparesis and homonymous hemianopsia. His jacksonian convulsions had been present since his birth by a prolonged, instrumental delivery. A ventriculographic study showed an enormous, somewhat localized dilatation of the body of the right lateral cerebral ventricle. The roof of this portion of the ventricle consisted of a thin, vascular, transparent, gelatinous structure. When it was opened the markedly hypertrophied choroid plexus floated. The choroid plexus was removed from the lateral ventricle and only the dura mater remained to cover the cortical defect. Surprisingly, the hemiparesis disappeared and for six years he has had no attacks.

In contrast are the remaining four patients in this group, all of whom had a localized area of cystic degeneration of the cortex and in three of whom there were overlying defects in the skull. Resection of the area of obvious gross pathologic change has in no way modified the course of the convulsive seizures. In each of these patients encephalographic studies

clearly demonstrated the mechanical defect in the ventricular system and subarachnoid spaces.

There remain five patients, all of whom had localized jacksonian attacks, with definite motor weakness and reflex changes following their convulsions. None of these patients gave a history of trauma at or following birth and there were no evidences of congenital defects, but in each there was a clear history of an infectious illness followed by the apparent residue of an acute or subacute encephalitis.

Here again the dubious character of the clinical results of operative interference is obvious. In one patient whose right-sided convulsions began at the age of 13, in whom evidences of paresis were present and who was in a status epilepticus, surgical exposure of the brain revealed generalized increase in the cortical vascularity and evident thickening of the arachnoid. The operation was followed by an entire freedom from seizures for seven years. It is difficult to attribute such a clinical result to a surgical procedure which amounted only to inspection of the brain. It is still harder to explain why the patient has recently had a recurrence of similar attacks.

In three other patients with a similar clinical story and with almost identical findings at operation, no such false hopes of a cure were raised. Their attacks continued unchanged in frequency or character in comparison with their preoperative condition.

In contrast, the remaining patient in this group presented encephalographic evidence of a dilated ventricle, its roof pulled toward the scarred and gliotic cortex, which had the consistency of a piece of gristle. One might justifiably believe that resection of this area, with demonstrable return of the ventricular system to the midline, would be followed by improvement, but such was not the case. Within a year the ventricular roof and lateral wall were again pulled outward by a contracting cicatrix, and at no time were the convulsions materially altered.

The recitation of these experiences in the surgical treatment of epileptiform seizures emphasizes to us the striking complexity of the entire mechanism of the convulsive disorders, and we have dealt only with those patients who have shown some concrete evidence that they might be benefitted by a surgical procedure. It is obvious that at least one factor, as yet ill-defined and comparatively unknown, must play an important rôle in the determination of successful results and failures. This factor of the so called "epileptic constitution" must be studied in these patients in whom surgical indications appear more or less definite, but first there must be agreement upon just what such a term implies.

Like some other fields in neurologic surgery this problem remains for solution. It cannot be dismissed as being completely unfruitful, but neither should operations be undertaken except under conditions which approximate those of the experimental physiologic laboratory as closely as is possible. Certain it is that freedom from attacks in patients three, five or even seven years after operation cannot be presented as evidence of the complete cure

of their attacks, but the accumulation of accurately reported data may eventually lead to the establishment of surgical indications and may provide a valuable link in the solution of an intricate neurologic problem ages old.

REFERENCES

[1] Pollock, L. J., and Davis, L.: Experimental Convulsions. Arch. Neurol. and Psychiat., vol. 20, p. 756, 1928.

[2] Penfield, W.: Diencephalic Autonomic Epilepsy. Arch. Neurol. and Psychiat., vol. 22, p. 358, 1929.

[3] Foerster, O.: Encephalographische Erfahrungen. Zeit. ges. Neur. u. Psych., vol. 94, p. 512, 1924.

[4] Foerster, O., and Penfield, W.: The Structural Basis of Traumatic Epilepsy and Results of Radical Operation. Epilepsy and the Convulsive State, p. 587. Williams and Wilkins Co., 1931.

[5] Penfield, W.: The Mechanism of Cicatricial Contraction in the Brain. Brain, vol. 50, p. 499, 1927.

CARCINOMA OF THE BREAST

A STUDY OF THE FIVE YEAR END-RESULTS

WILLIAM PERRIN NICOLSON, M.D.

ATLANTA, GA.

AND

MAXWELL D. BERMAN, M.D.

JACKSON, MISS.

FROM THE STEINER CANCER CLINIC, ATLANTA, GA.

THE subject of carcinoma of the breast has been so recently, thoroughly and adequately presented before this association by our late Fellow, Dr. Burton Lee, in 1932, and by our former president, Doctor Trout, in 1933, that no attempt will be made here to present an exhaustive treatise on the subject. This will be merely a presentation of certain facts and figures obtained from the observation and study of such cases occurring at Steiner Clinic from the time of its inception in September, 1924, to July, 1930. During these first years the material available was naturally much less than it is today, and the histories were frequently inadequate, making a complete study impossible.

During the period under consideration, 261 cases of carcinoma of the breast were admitted for treatment. Many of these had been operated upon elsewhere, and had recurrences, or were too far advanced to receive anything but palliative treatment.

We used the standard of operability laid down by Lee, namely, "A case is '*operable*' when a patient has a tumor of the breast that is not fixed to the chest wall, with or without invasion of the axillary lymph nodes. . . . If there are wide multiple cutaneous nodules around the original tumor site, if there is a swollen or painful arm with extensive axillary metastases, if the supraclavicular nodes are invaded, or if there are distant metastases to the chest or bones, the case should be considered '*inoperable*' . . ." Lee finished the sentence . . . "and radical operation should be withheld." We are not in accord with this last statement, and in a previous paper before this association one of us endeavored to show that frequently a radical operation, performed as a palliative procedure, would leave the patient more comfortable than she would have been without it. Some apparently hopeless cases have lived in comfort for many years. We feel that the primary standard of operability should be used merely in statistical studies, so that a fair comparison can be drawn between various series of cases and end-results evaluated. Many of the cases which are not primarily operable should be given the benefit of a radical surgical procedure. Consideration of one's personal statistics and records should never jeopardize the best interests of the patient. Employing the above standard, there were 74 "*operable*" cases.

First Symptoms in Cancer of the Breast.—As previously mentioned, complete data were frequently not available. Many cases had been operated upon elsewhere; in many, the records were incomplete and in others the patient had been "lost" from our follow up. In the following observations the number of cases comprising each group is noted; all cases in which the data were available were included. There was no selection. The first symptom occurring in 238 cases is detailed in Table I.

TABLE I

THE FIRST SYMPTOMS NOTED IN 238 CASES

First Symptom	All Cases		Primarily Operable		5 Yr. Cures. All Cases		5 Yr. Cures. Prim. Op.	
	%	No.	%	No.	%	No.	%	No.
Lump	75.21	179	26.25	47	8.38	15	31.91	15
Pain	8.	19	31.6	6	10.52	2	33.33	2
Lump in axilla	2.52	6	50.	3	16.67	1	33.33	1
Tenderness of, or Itching about nipple	2.52	6	50.	3	33.33	2	66.67	2
Ulceration of, or crust over nipple	2.10	5	60.	3	20.	1	33.33	1
Enlargement of breast	2.10	5	20.	1	0	0	0	0
Bleeding from nipple (lump?)	1.68	4	50.	2	50.	2	100.	2
Red area, or tender lump, or red nodule	1.68	4	75.	3	0	0	0	0
Burning or stinging sensation in breast	1.68	4	25.	1	25.	1	100.	1
Lump in supraclavicular region	.84	2	0	0	0	0	0	0
Wrinkle under breast	.42	1	100.	1	0	0	0	0
Sinking in of breast	.42	1	100.	1	0	0	0	0
Pain in lower back	.42	1	0	0	0	0	0	0
Swelling of arm	.42	1	0	0	0	0	0	0

These coincide strikingly with the figures given by Lee from an analysis of 1,000 breast malignancies. Consideration of the percentage of cases which were primarily operable, as well as the five year results as shown in Table I, bear out Lee's further observation that certain symptoms are more unfavorable than others and that these are doomed from the outset.

In our series the operations were performed by a number of surgeons, but in most cases were by one of our staff. As a general rule, a definite routine was carried out, namely, a preoperative cycle of high voltage roentgen therapy treatments, a suberythema dose being administered through four or five ports, followed by a radical mastectomy in from ten days to three weeks after the last treatment. A postoperative cycle was given 60 days after the preoperative treatments, and in the case of a radiation sensitive tumor, a second postoperative cycle was given two to three months later.

Subsequent radiation, either from radium or roentgen ray, was used where indicated in the individual case.

Occurrence by Age Groups.—The following table shows the occurrence of cases (based on 261 cases) by age groups (Table II), (Charts 1, 2, 3 and 4).

TABLE II

OCCURRENCE BY AGE GROUPS IN 262 CASES

Age	All Cases	Primarily Operable		5 Yr. Cures. All Cases		5 Yr. Cures. Prim. Op.	
		No.	%	No.	%	No.	%
26–30	5	1	20	1	20	1	100
31–35	8	4	50	1	12.5	1	25
36–40	31	9	29	3	9.6	3	33.33
41–45	38	11	23.7	6	15.8	6	55.55
46–50	36	10	27.77	1	2.74	1	10
51–55	43	17	40	6	11.6	6	35.35
56–60	36	8	22.22	3	8.33	3	37.50
61–65	21	9	42.85	5	23.8	5	55.55
66–70	17	5	29.3	3	17.6	3	60
71–75	9	2	22.22	0	0	0	0
76–80	7	0	0	0	0	0	0
81–85	1	0	0	0	0	0	0

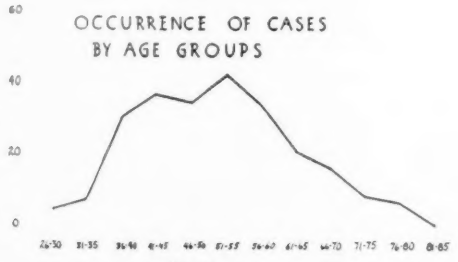


CHART 1.

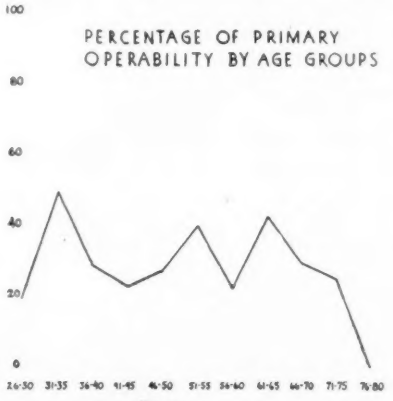


CHART 2.

It will be noted that the greatest number of cases occurred between 41 and 60, and of any five year period from 51–55. It is rather surprising to find such a high percentage of five year cures in the youngest group (Charts 3 and 4), but attention is called to the fact that there was only one primarily operable case in the 26–30 group, and that one happened to be free of symptoms at the end of five years. After that the percentage is below the series average until the 41–45 group is reached. Then there is an unexplainable drop to 10 per cent between 46–50. The percentage of cases which were primarily operable runs about as would have been expected, except for the unexplainable drop in the 56–60 group.

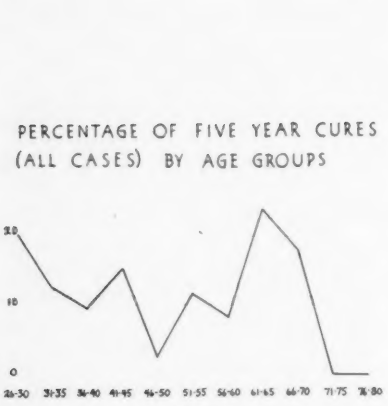


CHART 3.

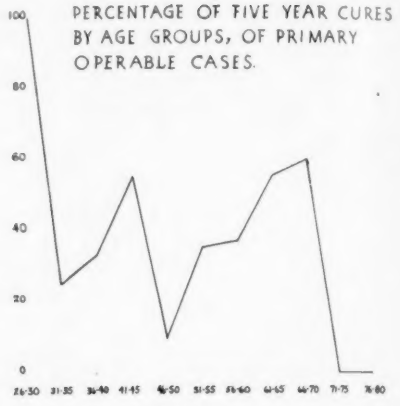


CHART 4.

Sex.—In the primarily operable group, there was no case in a man, and but one male case in the whole series. This is a smaller incidence than is generally found.

TABLE III

MARITAL STATE

	Percentage	Percentage Primarily Operable	% 5 Yr. Cures
Single	14	31.4	11.11
Widowed	36.4	29.6	14.2
Married	49.6	33	13.7

Marital State.—Table III, and Chart 5, show the marital state, the primary operability in each, as well as the five year cures. The primary operability is practically constant, but in the single persons there was a smaller percentage of five year cures.

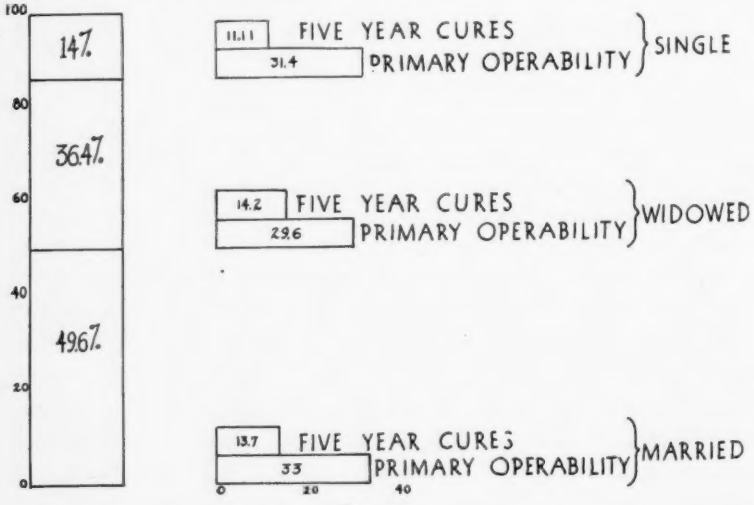


CHART 5.

Effect of Pregnancies.—Table IV, and Chart 6, show the division of cases according to the number of lactations (205 cases).

TABLE IV

EFFECT OF LACTATIONS IN 205 CASES

Number of Lactations	% of Cases	Primarily Operable		5 Yr. Cures. All Cases		5 Yr. Cures. Prim. Op.	
		No.	%	No.	%	No.	%
0	30	17	28.8	7	11.86	7	41.2
1	14	10	35.7	3	10.7	10	30
2	13.5	11	40.74	7	26	7	63.63
3	16.5	10	30.30	7	21.21	7	70
4	8.5	4	23.5	2	12	2	50
5	6	5	41.66	2	16.66	2	40
6	5	4	44.44	2	22.22	2	50
7	1.5	0	0	0	0	0	0
8	2	1	25	1	25	1	100
9	2.5	3	60	1	20	1	33.33
10	2	0	0	0	0	0	0
11	0.5	1	100	1	100	1	100

Note that no case had seven lactations—and such a small percentage had more than seven that the oscillation of the graph in the upper numbers is not reliable. A larger percentage of cases occurred in the nulliparous breast than in any one group, but there were over three times as many in those which had lactated. The primary operability seems to increase with two or

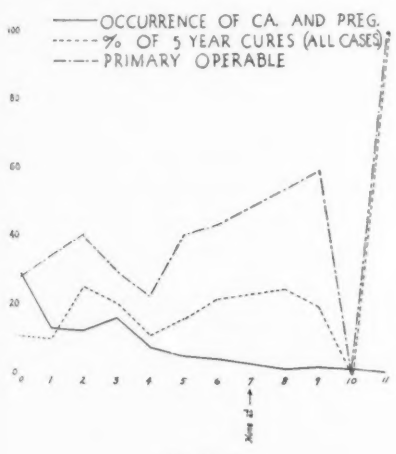


CHART 6.

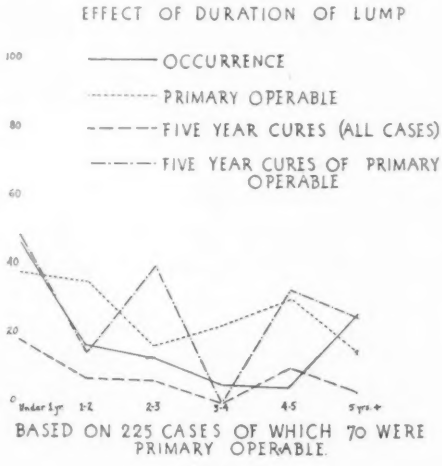


CHART 7.

more pregnancies, as well as the percentage of five year cures. From this it might well be deduced that when a breast has performed the function for which it was intended there is less chance of a carcinoma being inoperable and a greater chance of cure. It logically follows that if for any reason a

lactation is interrupted, the breast should be adequately and persistently emptied with a breast pump. At each ovulation the breast undergoes a change in preparation for lactation. Following failure of the ovum to be fertilized, the breast should theoretically return to normal, but practically this does not always take place. Bagg and Adair showed that carcinomata could be produced in rats' breasts by merely blocking the ducts and allowing the animal to continue to breed.

Effect of Duration of the Disease.—Based upon 225 cases in which information is available, Table V, and Chart 7 present the effect of the duration of the disease. All cases in which the disease has been present for more than five years before admission have been grouped together. Otherwise, they are classified by years.

TABLE V

EFFECT OF DURATION OF DISEASE IN 225 CASES

Duration of Disease	% of Cases	Primarily Operable		5 Yr. Cures. All Cases		5 Yr. Cures. Prim. Op.	
		No.	%	No.	%	No.	%
Less 1 yr.	47.15	41	38.31	20	18.69	20	48.78
1–2 yrs.	17.55	14	36	3	7.7	3	14.3
2–3 yrs.	13.33	5	16.67	2	6.67	2	40
3–4 yrs.	5.78	3	23	0	0	0	0
4–5 yrs.	4.44	3	30	1	10	1	33.33
Over 5 yrs.	11.55	4	15.4	1	3.8	1	25

The primary operability decreases until the disease has existed longer than three years, and then shows a progressive increase for two years. Could this be due to the probably low malignancy of any growth which would persist longer than three years?

Clinical Versus Pathologic Grading as an Aid in Prognosis.—Trout quotes many authorities and proves rather conclusively that a classification based on clinical evidence is of more value from a standpoint of prognosis than is one graded histologically without any consideration or knowledge of the clinical history.

In the present series the tumors had not, as a rule, been graded pathologically. The names and numbers of the primarily operable patients were given to our pathologist, Dr. Everett L. Bishop, and he was asked to grade the slides histologically, without any knowledge of the clinical histories (Chart 8).

The same cases were graded clinically according to the Clinical Index of Malignancy (Lee) (Chart 9).

It is interesting to note that the percentage of cases occurring in the corresponding grades is strikingly similar, but in the histologic grades the

five year cures are not reduced as much as one would expect in the Grade III cases. It has been suggested that this might be because these tumors are more sensitive to radiation. This does not explain so great a disparity.

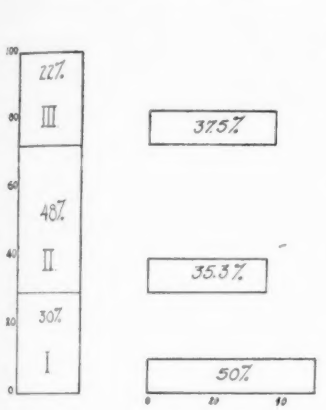


CHART 8.
Pathologic grading.

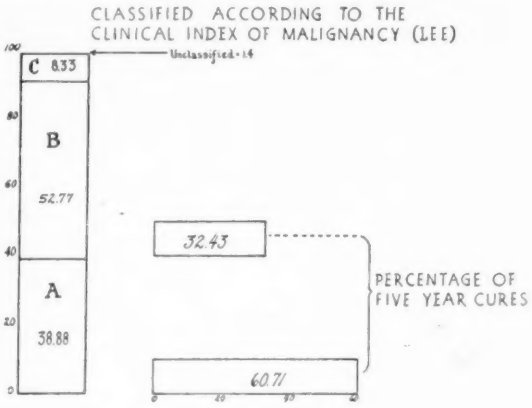


CHART 9.
Clinical grading.

Neither system is infallible. There were notable exceptions in each. For example, one case graded A and III is living without evidence of disease over nine years, while another graded A and I died from her cancer in nine months. In at least 11 cases, of which the above two represent the extremes, either one or both systems of prognostication failed utterly. In most cases, however, in our series, one is justified in feeling that the Clinical Index of Malignancy gives a reliable indication of the probable outcome, and is more dependable than a pure histologic classification. The pathologist, as one member of the "cancer team," should have sufficient clinical data to allow him to more accurately grade the tumor.

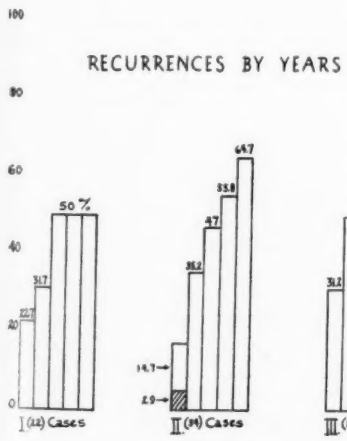


CHART 10.
Pathologic grading.

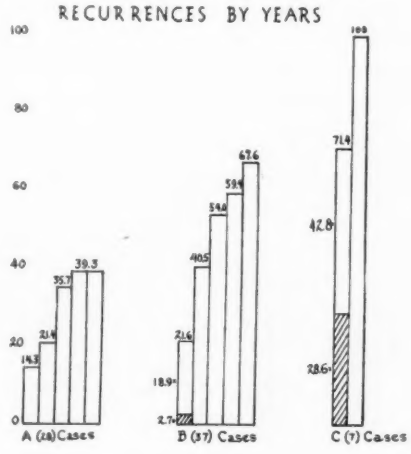


CHART 11.
Clinical grading.

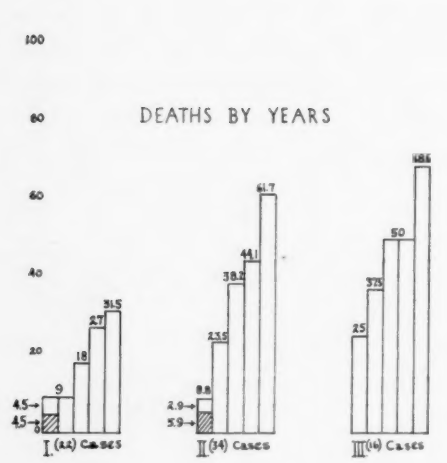


CHART 12.
Pathologic grading.

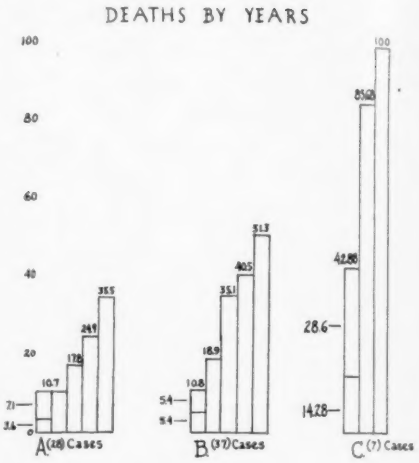


CHART 13.
Clinical grading.

Location of Tumors in the Breast.—In the 61 cases of the primary operable group in which the location of the tumor was stated, and in which the whole breast was not involved, we find 54.10 per cent of the cases occurring in the left breast. Chart 14 indicates the percentage of cases occurring in each part of the breast, and Chart 15 the five year cures by locations. The latter is based on too small a number of cases in each location to justify many definite conclusions, but the overwhelming preponderance of the tumors in the upper half of the breast is certainly significant. Why should so many more tumors occur in the upper portion of the breast? Could it not be explained by the imperfect lymphatic and other drainage from the upper portion of the gland, due in turn to the poor support, natural or artificial, of the breast? Is this another penalty exacted because we now walk on two legs? Trout refers to the absence of carcinoma in the udder of the cow, and ascribes this to her frequent and continuous use of the breast for its physiologic purpose. Might not the lowly cow, in spite of the frequent lactations, experience a malignancy in her organ of lactation more frequently if she

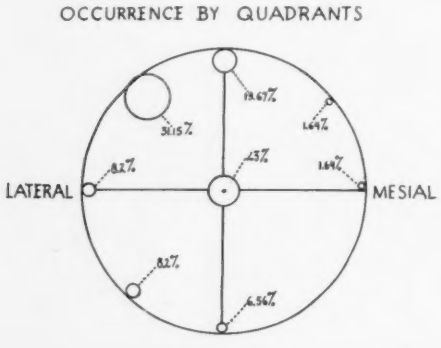


CHART 14.

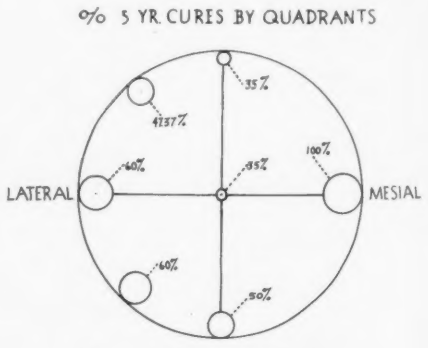


CHART 15.

walked on but two legs, thus interfering with the drainage? The former generation broke down the support of the breast, and interfered with its circulation, by wearing such tight brassieres, and the present woman is doing almost as much harm by wearing practically no support at all. It is encouraging to note the prominence of the breast in the modern styles—the brassiere now being chosen which accentuates the breasts. Though many of these need improving, they are much better than were formerly worn. We feel that the use of the proper type, supporting, but not constricting, brassiere will go a long way toward reducing breast lesions, especially malignant ones. It should be chosen not with an idea of concealing the breast, but rather of supporting it. In extreme cases some form of breast lifting, or plastic operation, is not only justified but also indicated.

Of the deaths, one occurred within a month of the operation, and was apparently not due to her carcinoma. No autopsy was obtained. Two others died, and autopsies proved there was no evidence of carcinoma of the breast but one of these did have carcinoma of the uterus. The one which had had a recurrence but who was now free of disease was not included. Seven were not traced, three of these were known to have recurrences, but four showed no evidence of disease when last seen, two (both graded B and II) having lived over four years. Should the one operative mortality and the two who died from an intercurrent disease be excluded, the percentage of five year cures would be raised to 38 per cent. If the four untraced ones which were free from disease when last seen, be omitted, it would be further raised to 40.3 per cent. Should these four untraced cases be included in the series as five year cures it would be still further increased to 46 per cent.

TABLE VI

RESULT AT END OF FIVE YEARS IN 74 PRIMARILY OPERABLE CASES

Dead	Living but with Recurrences	Died (Other Causes) Autopsy Proved	Living without Evidence of Disease
34 = 46%	3 = 4%	2 = 2.7%	27 = 36.48%

Lost Trace of but Known to Have Recurrence When Last Seen	Lost Trace of But Free of Disease	Had Recurrence But Now Shows No Disease
3 = 4%	4 = 5.4%	1 = 1.35%

Of the primary operable cases, 5 (6.7 per cent) had a bloody discharge from the nipple; 3 (4 per cent) had an erosion or crust over the nipple. Skin attachment was noted in 42 (56.7 per cent). In 33 (44.6 per cent) involvement of the axillary nodes was noted clinically; in 30 (40.5 per cent) it was noted pathologically. Primary skin grafts were utilized in 2 (2.7 per

cent). Of 40 cases in which no involvement of the axillary nodes was demonstrated, 19 (47.5 per cent) showed five year cures. In the whole series metastases to bones were noted in 35 (13.4 per cent) cases.

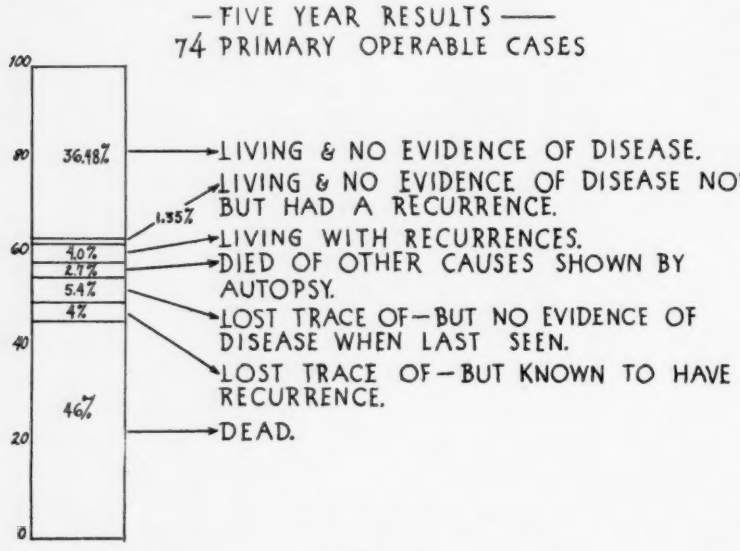


CHART 16.

The routine handling of breast malignancies during this period was, as previously stated, to have preoperative radiation, followed in ten days to three weeks by a radical mastectomy, and then one or more cycles of postoperative radiation. Because of the relatively large number of surgeons involved, this routine varied (Table VII).

TABLE VII

VARIABILITY OF PRE- AND POSTOPERATIVE TREATMENT

Preoperative radiation, radical mastectomy, postoperative radiation	13
Radical mastectomy and postoperative radiation	6
Excision of nodule, interstitial radium seed, roentgen radiation	2
Simple mastectomy, no radiation	1
Preoperative radiation and simple mastectomy	1
Simple mastectomy and postoperative radiation	1
Preoperative radiation, simple mastectomy, postoperative radiation	1
Radical mastectomy, postoperative radium	1
Preoperative radiation, radical mastectomy	1
	27

It should be noted that in six (22.22 per cent) cases no radical operation was performed and in two of these the tumor only was excised. We do not advocate the abolition of radical surgery, yet so many cases in the five year group who did not have it makes us think. After reporting six cases which had apparently been hopelessly inoperable, but which were living longer than 5 years, Wood says, "These cases have been reviewed to show that neither the histologic nor clinical grading of tumors can be relied upon for the correct

prognostication in the individual case, and that to obtain the best results in cancer therapy all growths, no matter how advanced, must be treated in a thorough and intelligent manner. The biology of the patient and the biology of the tumor, which he harbors, are the two great unknowns present in every case of cancer." But for these "two unknowns," the cases in which a radical procedure is indicated could be better differentiated. Until such time as they become "knowns," the safer procedure will be to give the patient the benefit of every possible means at our disposal.

CONCLUSIONS

(1) In more than three-fourths of the cases a lump is the first abnormality noted by the patient. In one of every 12 cases pain is the first symptom. Certain symptoms, such as painful swelling of arm, diffuse involvement of breast, pain in the lower back, or lump in the supraclavicular region, are more unfavorable than others.

(2) That cancer of the breast may occur earlier than the usually considered "cancer age"—five of this series occurring between 26–30 and eight between 31–35. The peak is reached in the five year period from 51–55.

(3) Only 14 per cent occurred in single persons but in these the percentage of five year cures was less than in those married.

(4) The primary operability of the tumor decreases in proportion to the length of time the lesion has existed, up to the three year period, when there begins a slight increase. The five year cures show a similar increase in the cases which have existed for many years.

(5) Two indices available to determine the prognosis—one, The Clinical Index of Malignancy (Lee), and the other, Histologic Grading of the Tumors. The former seems to be the more reliable, especially in the more hopeless cases.

(6) Eighty-five per cent of primary operable cases occurred in the central or upper portions of the breast. It is thought this may possibly be due to disturbance of circulation due to styles, *etc.*

(7) Thirty-six point forty-eight per cent of primary operable cases are living and free from malignant disease at end of five years.

DISCUSSION.—DR. HUGH H. TROUT (Roanoke, Va.).—There are three points concerning cancer of the breast about which I wish to speak. First, we still believe preoperative and postoperative irradiation is of value. We believe in it thoroughly, and we believe also thoroughly that it is now carrying with it a decided increased danger. There is apparently an increasing tendency throughout the country to do "sloppy" operations and to depend too much on postoperative irradiation. Some are advocating a simple mastectomy for carcinoma of the breast, and depending on irradiation to take care of the nodes that might be involved. We feel that this is distinctly harmful. With the new type of roentgen therapy machines and with the Coutard technic which gives large doses very quickly, I make the prediction that we will see some terrific burns. We have already seen them. However, these burns can be, and are, being prevented, by the more expert radiologists. I am sure that

ultimately these catastrophes will not occur even in the hands of the average technician when the control of these very high voltage machines, and the results of exposure to the same, are better understood.

Radium, unless properly screened, will cause necrosis of the cartilage of the ribs. You will recall that radium is placed in the area supplied by the internal mammary vessels, as well as in the axilla, and therefore, such cartilages are in close proximity to it. Necrosis of the cartilage is one of the most difficult problems with which I am familiar, and when it occurs frequently means the excision of the cartilage.

In visiting various clinics I obtain the impression that the Clinical Index of Malignancy as advocated by Lee and Stubenbord is gaining in popularity, as a means of estimating the prognosis. However, we still feel that both the clinical and the histologic methods should be employed jointly.

I think if we will all study the histories of our cases more carefully we will find a group of symptoms that apparently existed before the carcinoma developed. Along this line, I have in mind the question of painful breast associated with menstruation. Some of these cases can be relieved by the administration of the proper hormone, but, frankly, I do not know which are the proper hormones. There evidently is some definite association between mastitis and hormones.

Apparently the increase in carcinoma of the breast started about the same time as the theory that babies should be removed from the breast early, and that the breasts should not be pumped but allowed to "dry up."

I am hopeful that now with the increased use of a brassiere, that supports the breast instead of pulling it down, it may be of some benefit, for you will recall that carcinoma of the breast is most frequent in the upper, outer quadrant—the site where there is most obstruction due to traction of the old binder brassiere and corsets.

The correction of pelvic conditions may help. We have all seen pelvic conditions that apparently increased the tendency to pain in the breast, which might be a forerunner of carcinoma.

If we study the histories of our cases more carefully we might reach some conclusion as to how to decrease the percentage of occurrence of carcinoma of the breast. At least we might find some condition which will justify earlier and more hopeful operations. Frankly, I am afraid we have, with complete radical operations, plus irradiation, reached the limit of operative cure of carcinoma of the breast, and that now the hope of improvement in this situation rests in the education of ourselves, as well as the laity, in the proper evaluation of symptoms and conditions that might be precancerous.

Dr. Frank P. Strickler (Louisville, Ky.).—We see cases of carcinoma of the breast that recur after 20 years. They are being reported more and more frequently. It has occurred to me that we may be using the wrong terminology when we say "cured." We might use "arrested" instead of cured, and then we might not have to take it back. We have heard about carcinoma being caused in the female breast by hormones, lactation, the number of children and so on, but what about the number of carcinomata that occur in the male breast? I think we cannot ignore these entirely. They have the same type of growth and the same type of tissue. I have seen several of these cases and certainly the same etiologic factors do not obtain as in the female. There must be some other reason for carcinoma that we have not considered.

Dr. Carrington Williams (Richmond, Va.).—I wish to mention a case that was operated upon 43 years ago by Dr. Hunter McGuire. The breast was completely removed, but the pectoral muscles apparently were not disturbed. There are no hospital records that we can find but the patient was told that she had carcinoma of the breast. Within the last few months she has had an abdominal enlargement and examination showed that the liver was definitely enlarged and there were several nodules on the liver; it seems reasonable therefore, to believe that she had carcinoma of the breast in 1893 and that she now has carcinoma of the liver. This would tend to confirm the report of Doctors Lewis and Rienhoff, which was the most dismal that I have ever heard. They had followed their cases carefully and some had recurrences after 40 years. I think this report of Doctor Nicolson's is very interesting, and in time such records will help us to find out what really to expect in carcinoma of the breast.

Dr. William Perrin Nicolson (closing).—I agree fully with Doctor Trout. We have not been using the Coutard technic in operable cases. Our routine calls for a cycle of four treatments through relatively small ports, including the breast, tumor and axilla.

I did not mean to say that I considered these cases which were free of disease at the end of five years were "cured." I doubt if a case of carcinoma of the breast can ever be considered as "cured," in that they may occur even after an incredible number of years, as in the case Doctor Williams reports. Every case should have periodic postoperative examinations as long as she lives.

THE ULTIMATE PHASE OF LIFE AS IT RELATES TO WOUNDS OF THE HEART

WALLER O. BULLOCK, M.D.

LEXINGTON, KY.

On July 16, 1927, a male, aged 20, was admitted to St. Joseph's Hospital, having been shot some 20 minutes previously. The patient was in shock, almost pulseless, but conscious, with a bullet wound in the third left interspace three-fourths of an inch from the sternal border. The physical signs indicated a wound in the heart. Within 30 minutes after the accident he was operated upon under light ether anesthesia. An incision was made through the cartilages to the left of the sternum following the Kocher procedure. Before reaching the pericardium the patient's heart action ceased entirely. The writer remarked, "I have never before operated upon a heart wound, let's just go ahead and complete the operation on the cadaver." So deliberately, and without haste, the chest was opened widely, cutting through the pleura as well as the pericardium, releasing the clot and fluid blood from the pericardial sac. The heart was motionless in the back of the cavity with dark fluid blood pouring in a steady stream from an opening in the right ventricle; the wound was a long tangential one tearing through into the right ventricle and making a trough in the muscle of the left ventricle and the interventricular septum about two inches from the apex. Holding the heart in the left hand a suture of chromic catgut was placed to close the opening. After placing the second suture a slight crawling movement of the heart was felt. This was repeated again after the next suture had been introduced, a little stronger than before and again and again until by the time the wound was completely closed the heart was beating normally. The patient left the table in fair condition.

Following the operation he developed a purulent pericarditis which was aspirated several times and an empyema of the left pleural cavity, which had to be drained, but he finally recovered and is at the present time in good health.

The massage together with the needle pricks which the heart received during the procedure were sufficient to set it in motion again. The interval of complete cessation of heart beat was probably about ten minutes. No effort was made at artificial respiration as the patient was supposedly dead.

The case itself, besides contributing to the writer a tremendous reaction from having a supposed cadaver come to life while holding the heart in his hand, was regarded merely as one of those extraordinary happenings that occurs occasionally in the experience of every surgeon. Within the past year, however, attention was called to the work of Doctor Cornish of California on "Resuscitation of Dogs." Hymans' researches on the same subject with special reference to restarting the stopped heart with his pacemaker were reviewed. Subsequent investigation of the entire subject of resuscitation in the Library of the Surgeon General at Washington was undertaken and justifies the following statements.

That there exists an ultimate phase of life after the heart and lungs cease to function in which it is possible to restart these functions.

That the duration of this ultimate phase has not been definitely deter-

mined and that it probably varies with different individuals, with age, and with preceding condition of vigor.

That the cells of the centers of circulation and respiration withstand deprivation of blood (oxygen) longer than do the cells of the cortex.

That deprivation of blood (oxygen) from the cortical cells beyond a certain point, even if life is restored, results in harmful changes in personality.

That the actual time in minutes in which human life has been restored without injury to the cortical cells after the heart and lungs have ceased to function has varied in the reported cases from eight to 30 minutes.

With this light upon the case here reported it is evident that the result was not just an accident but was one that may be reasonably expected under similar conditions.

Death from heart wounds is due to hemorrhage or heart compression. Where the pericardial tear is large, death occurs rapidly from bleeding into the pericardial sac. When it is small death is due to a gradual compression of the heart and the process consumes a longer time. It is this latter class that has furnished the favorable surgical material.

Since Rehn first reported in 1897 the successful suture of a heart wound, the literature on the subject has been steadily increasing. No fewer than 44 articles on this subject are cited in the Index Medicus during the past two years. In a review of this literature the reader often met with the expression "the patient died on the table and the operation was discontinued" or "patient expired before anything could be done." It is the purpose of this paper to draw attention to the fact that there still remains the ultimate phase of life; that the patient is at first not really dead, even though his heart has ceased to beat and air no longer enters his lungs; that the surgeon still has time to do what should be done. It may even be shown by further experience that on account of the rapidity and ease of its performance and on account of the lessened danger of exsanguination, the ultimate phase may provide better figures from the standpoint of recovery than the penultimate or just preceding period. The fact that the patient is in the stage commonly called the "article of death" is no contraindication to operation.

And, finally, it is the author's opinion that if all patients with heart wounds dying in the ambulance, or shortly after admission, are sent not to the morgue but to the operating room and operated upon immediately, some of them will recover.

SUPPURATIVE PERICARDITIS

LATE RESULTS AND METHODS OF DRAINAGE

ARTHUR M. SHIPLEY, M.D.

BALTIMORE, MD.

FROM THE UNIVERSITY HOSPITAL CLINIC, BALTIMORE, MD.

DOCTOR GEORGE H. BUNCH[1], in 1934, presented four personal cases of suppurative pericarditis before the Southern Surgical Association. Doctor Nathan Winslow and the writer,[2, 3] collected 227 reported cases of pyopericardium up to January 1, 1934, 12 of which were personal experiences.

After having operated upon 12 cases of pyopericardium with seven recoveries and five deaths, I am discussing this condition with two objectives in mind, one of which is to report the present condition of the patients who recovered, and the other is to bring up the question of adequate drainage.

There is a widespread belief that most patients operated upon successfully for pyopericardium are thereafter seriously crippled by adhesive pericarditis. One would naturally expect such a result. In 1929, Horine[4] reported his findings on dogs whose pericardial sacs were infected by injecting either pneumococci or staphylococci into them, then dropping them back into the thorax and closing the wound in the chest. In all of these dogs the postmortem findings were essentially identical, adhesive pericarditis with collections of pus in the auricular areas or lateral recesses, complicated by extensive infection of neighboring structures.

Beck and Moore[5] made a transpleural approach under positive pressure anesthesia and injected the pericardium, afterward closing the chest wall. Some of these dogs recovered and a number died in a few days.

When the condition of the pericardium at operation is considered and when the effects of drainage within a serous cavity are observed, widespread adhesions between the two layers of the pericardium more or less obliterating the pericardial space seem inescapable. And when the very active heart is visualized, in repeated systole and diastole, one is surprised that it continues to adequately perform its work in the presence of such pathology. But there is abundant proof that such a heart does continue to do its work without clinical evidences of any serious lessening of its ability to function.

In many of the cases collected by us, no statement was made beyond the fact of operative recovery and no conclusions can be drawn from these. Fortunately, however, 39 cases are available in the literature, in whom at least one year had elapsed since the pericardiotomy. Thirty-five of these patients were alive and well at the time of the report, with their cardiac boundaries within normal limits. One was alive but had adhesive pericarditis, and his death was momentarily expected. Of the three remaining one had died three years after the pericardiotomy, but the cause of death was not known; one

had died of an abscess of the brain a year later and one of an adhesive pericarditis.

I have been able to keep in touch with six of the seven recovered cases in my own series. The one patient whom we have been unable to locate was a girl, 12 years old, who was operated upon January 25, 1927. She had a stormy convalescence for two weeks and then made a rapid recovery and was discharged March 16. She was followed for several months, but was lost sight of and has not been examined since.

All of the remaining six were young persons—4, 9, 17, 18, 19 and 29 years old respectively. Only one had an associated empyema and he was the oldest. In five of the seven the pericarditis complicated pneumonia; in one it followed scarlet fever, and in the youngest, 4 years old, tonsillitis and otitis media with arthritis preceded the pericarditis. This patient was operated upon in June, 1924. Paracentesis pericardii was performed before operation and a pneumococcus, of no fixed type, was grown from the serosanguinopurulent fluid. He had a stormy and prolonged convalescence. After all evidence of acute infection had subsided, and his drainage wound had healed, he continued with the symptoms of serious cardiac damage for several months accompanied by anasarca, ascites and dyspnea. His peritoneal cavity was tapped several times. The fluid was clear and showed no growth. His heart was enlarged but no signs of mediastino-pericarditis could be made out. Whether adhesive pericarditis or myocardial damage was playing the major rôle we were unable to say. If the pericardial effusion had not been purulent and associated with a pneumococcus, we would have felt that we had performed pericardiotomy for pericardial effusion, associated with rheumatic fever. I have seen the boy many times since. He is now nearly 16 and is large for his age, robust, active and with no evident disability.

Another of these patients had a slow convalescence. She was 19 when operated upon in 1929. Her pericarditis complicated pneumonia. The pus was thin, under pressure, and gushed from the incision. Two small tubes were placed behind the heart for drainage and sutured to the skin. For several days her condition was desperate and on the fourth day irrigation was begun. Her condition improved, but even two months after operative recovery she had considerable shortness of breath when she was very active. Again we could find no evidence of mediastinopericarditis or of constricting pericarditis. She has now completely recovered clinically, and is not conscious of any disability.

In another patient there was an unusual complication. This was a girl, 9 years old. Her pericardium was drained with two small rubber tubes. She did well for two weeks and then had fever with hurried breathing and a fast pulse. Roentgenography showed a suspicious enlargement of the cardiopericardial shadow to the left and in the region of the base of the heart. A walled-off pocket was suspected and found by removing the drains and following with the finger along the drainage tract, posterior and lateral to the left ventricle. This patient is now grown and married. She is clinically well.

A boy, 17 years old, was operated upon in 1930. He had a history of valvular heart disease preceding his illness, which complicated scarlet fever. His pericarditis was purulent and a hemolytic staphylococcus was recovered. He made a rapid and satisfactory recovery except that he developed thrombophlebitis in one leg. I saw him recently. He is now active. He still has his valvular lesion, but his heart is compensated. When he has a long, hard day, he has some swelling in the leg, the result of the phlebitis. His future is, at best, uncertain but his circulatory system is carrying on in spite of a triple handicap.

The 18-year-old boy was operated upon in 1925. After recovery he passed the physical examination for admission to the Naval Academy, but was rejected because of the history of suppurative pericarditis. He reports himself in good health. The last case was operated upon two years ago. He writes that he is well. Dr. Harvey Stone operated upon a second case several years ago with recovery. This patient has no clinical evidence of disability.

Of the cases that recovered all were operated upon early. Most of them were seen in consultation with Dr. M. C. Pincoffs. Of the five fatal cases, only one was a late case, he was also the oldest patient. That defective drainage played very little part in the fatal outcome in this series is suggested by the fact that the time factor between pericardiotomy and death was short in all five cases; a few hours, the next day, 26 hours, 12 hours, a few hours, being the respective notes attesting to this time period.

It is easy to understand why patients operated upon for suppurative pericarditis should succumb to early complications and infection and later to adhesive pericarditis. In only one of our cases was any attempt made to suture the edges of the pericardium to the sides of the incision in the chest wall. The middle portion of the lower mediastinum is occupied by the heart and pericardium. The outer layer of the pericardium is attached to the heart and to the middle of the diaphragm over a considerable area surrounding the quadrate opening through which the inferior vena cava passes from abdomen to right auricle. With this exception the heart lies free in the pericardial sac which extends upward onto the great vessels for a distance averaging 5 cm. A part of the outer layers of the pleurae are in intimate contact with the outer layer of the pericardium, but these structures are not adherent.

One would expect pus to gravitate from the opening in the pericardium downward along its outer surface and set up some reaction between itself and the enveloping pleurae. One would also expect some evidence of posterior mediastinitis. Postmortem findings have not been very accurate in most instances. The usual operation for pyopericardium is through the "triangle of safety" by removal of some part of the chest wall immediately in front of the left ventricle. This is the region in which crippling mediastino-pericarditis is seen and for which the Brauer operation is indicated, but I do not remember any report of this operation having been performed for mediastino-pericarditis following drainage of the pericardium.

On the other hand, there is no reason why the pericardium, thickened and

inflamed and adherent as the result of infection followed by drainage, should not undergo fibrosis and contraction bringing about constricting pericarditis calling for pericardiectomy, but I do not recall that any of the 37 pericardiectomies collected by Churchill[6] were undertaken to liberate the heart in any patient where the concretio pericardii was preceded by pericardiotomy for suppurative pericarditis.

There are many ways in which chronic adhesive pericarditis shows itself anatomically and these different phases may be visualized under four heads. First, adhesions between the inflamed pleura and the outer layer of the parietal pericardium, seen commonly in unilateral fibroid phthisis where the heart and pericardium are pulled somewhat into the diseased side of the chest. Second, mediastino-pericarditis. Third, constricting pericarditis, and fourth, adhesions between the two layers of the pericardium, more or less obliterating the pericardial sac, but without constriction and without the pericardium being adherent in turn to the unyielding chest wall. In this type of chronic pericarditis the heart is perhaps not seriously handicapped in its movements because under normal conditions the pericardium follows the heart closely. It is possible that many of the patients who are clinically free of cardiac disability following pericardiotomy belong in this last group and the individuals who are victims of cardiac disability following pericardiotomy may suffer from some combination of the different types of chronic pericarditis.

Method of Approach.—Emphasis had been laid by most operators on the "triangle of safety" and the approach through the chest wall has usually been either a left or right parasternal, a trans-sternal, a chondroxiphoid or some combination of these. In the last two cases operated upon by me a modified approach was made combining the trans-sternal with the chondroxiphoid. This is a low anterior approach and gives a wide exposure of the uncovered pericardium between the medial margins of the two pleurae, brings the margin of the left pleura into view and exposes the left internal mammary vessels just above the diaphragm. It is a low approach in the caudate cephalad axis of the body and allows a wide opening which permits exploration of the pericardial sac by the finger, which is an aid in estimating the extent and location of the effusion and the presence of adhesions which might lead to walling off of the exudate, and in placing the drains. In the dorsal-ventral axis, however, this is an anterior approach, and with the patient lying on his back the drainage is directly against gravity. The movements of the heart have some tendency to promote drainage and attempts have been made to improve it by changes in the position of the patient.

In the absence of adhesions, the apex of the heart is usually found in close proximity to the anterior layer of the parietal pericardium and most of the pus is posterior and lateral to the heart. When a part of the fluid escapes, the ventricles are apt to interfere with anterior drainage, unless drains can be maintained in position either posterior or lateral to the heart. This anterior approach has therefore been objected to, and has been found unsatisfactory at times, especially in instances of shaggy pericarditis where there are masses

of coagulated fibrin which repeatedly plug the drainage tract, or where there is a long standing effusion with a very large and adherent pericardium.

Posterior drainage has been considered and tried, but the difficulties and risks are very great. The vertebral column and the structures in the posterior mediastinum are in the way when attempting an extrapleural posterior approach and a paravertebral approach can be made only by going through the pleura and the lung, provided the risks of going through the posterior mediastinum are avoided. Both Truesdale[7] and Cottam[8] writing in the same year, 1933, criticize the anterior approach for the reasons mentioned above and each reports a different posterolateral approach. Ballance, in his Bradshaw lecture, discusses this problem because of his experience for three and one-half weeks with a posttraumatic case which finally succumbed. Cottam drained a case successfully by locating pus by paracentesis and with the needle in place followed it by removal of the right fifth costal cartilage, incising both layers of the pleura, which were adherent, and by burrowing backward, he opened the pericardium well away from the front. On introducing his finger he felt the heart anterior and to the left and inserted a large, soft rubber tube backward and upward until he met resistance. A number of objections may be raised to this procedure, but he succeeded in getting good drainage and his patient recovered.

Truesdale lost a patient in 1910, 16 days after anterior drainage, by approaching the pericardium through the left fourth and fifth chondral cartilages. In 1932, he saw another case of pyopericardium and against his better judgment again drained the pericardium through the region of the left fourth and fifth cartilages. When the pericardium was opened approximately 2,500 cc. of pus escaped under great tension, but the patient continued ill and drainage was greatly interfered with by clumps of fibrin. Twenty days after operation the patient became worse and after transfusion a horizontal incision was made in the left seventh interspace beginning at the nipple line and extending outward to the posterior axillary line. About two inches of the seventh and eighth ribs were resected. On opening the pleura the pericardium was seen resting on the diaphragm and the lung and pleura were adherent to it. It was necessary to dissect these overlying structures away before opening the pericardium. The pleural cavity was not opened. Through and through drainage was established and the patient promptly recovered.

Heuer[9] had the same experience. When he resected the left fifth costal cartilage he found the heart adherent to the parietal layer of the pericardium, and efforts to separate the adhesions were damaging to the heart muscle. The patient was placed on his side and a rib in the left posterior axillary line was resected; the pleurae and lung were found adherent and the pericardium was reached through a corridor in the lung and the patient recovered.

Loucks[10] reports an instance of suppurative pericarditis following a wound of the chest. Anterior drainage by left parasternal approach through the cartilage of the sixth rib was done. No drainage tubes were used. Drainage was not satisfactory and several days later a localized empyema was drained

after resection of the eighth rib posteriorly. Thirteen days after the anterior pericardiotomy the pericardium was drained posteriorly through the posterior opening made for draining the empyema. This patient recovered.

Moore[11] reported a left sided posterolateral drainage similar to that of Truesdale, and with the same findings as to distended pericardium and adherent lung and pleura. There had been no previous anterior pericardiotomy.

In all of these cases the pericardium was reached in the posterolateral plane and as far as the heart was concerned the drainage was from behind. The fortunate thing in each instance was that the free pleura was not opened and no pyopneumothorax resulted. In large, long-standing effusions anterior drainage may be insufficient and the chances are in favor of reaching the pericardium laterally without opening the pleural cavity. The above reports are a very valuable addition to our knowledge of the subject.

In early suppurative pericarditis, when the pus is still thin and before distention of the pericardium is marked and before adhesions between pericardium and pleura may have had time to form, any approach except through the "triangle of safety" is apt to open and infect the pleural cavity. Pyopericardium and pyopneumothorax in the same patient at the same time offers a bad prognosis.

CONCLUSIONS

All the patients that I have operated upon, except one, were relatively early cases. I believe that the low anterior approach is better than the higher parasternal one through the level of the fourth and fifth costal cartilages. Tube drainage with two small tubes, placed with the finger, behind the heart and fastened to the skin margins to prevent displacement will expedite drainage and irrigation may be a useful adjunct, provided fluid escapes from the pericardial sac as fast as it is introduced.

REFERENCES

[1] Bunch, George H.: Suppurative Pericarditis. Am. Jour. Surg., vol. 28, p. 613, April to June, 1935.

[2] Winslow, N., and Shipley, A. M.: Pericardiotomy for Pyopericardium. Arch. Surg., vol. 15, p. 317, September, 1927.

[3] Shipley, A.M., and Winslow, N: Purulent Pericarditis: Report of Five Cases in Which Treatment Was by Pericardiotomy and Review of Literature from April 30, 1927, to January 1, 1934. Arch. Surg., vol. 31, p. 375, September, 1935.

[4] Horine, C. F., and Shipley, A. M.: Experimental Pericarditis. Arch. Surg., vol. 18, pp. 386–402, January, 1929.

[5] Beck, C., and Moore, R.: Significance of Pericardium in Relation to Surgery of the Heart. Arch. Surg., vol. 11, p. 550, October, 1925.

[6] Churchill, Edward D.: Decortication of the Heart for Adhesive Pericarditis. Arch. Surg., p. 1455, December, 1929.

[7] Truesdale, P. E.: Low Pericardiotomy for Acute Suppurative Pericarditis. New England Jour. Med., vol. 208, p. 671, 1933 (2 cases).

[8] Cottam, G.: Suppurative Pericarditis: Description of a Case in Which Drainage Was Made Through New Approach. West. Jour. Surg., vol. 41, p. 87, 1933.

[9] Heuer, G. J., cited by Loucks: Arch. Surg., vol. 18, p. 859, March, 1929.
[10] Loucks, H. H.: Suppurative Pericarditis. Arch. Surg., vol. 18, p. 852, March, 1929.
[11] Moore, Richmond L.: Posterior Drainage in Suppurative Pericarditis. Arch Surg., vol. 102, p. 980, December, 1935.

DISCUSSION OF THE PAPERS OF DOCTORS BULLOCK AND SHIPLEY

DR. ALBERT O. SINGLETON (Galveston, Tex.).—Wounds of the heart usually occur at a time of day when the attending surgeon is not available.—Most commonly at night, when the younger surgeons in the hospital have the responsibility of caring for them.

I have been of the opinion that a plan should be formulated by which we may expedite the prompt care of these patients. This plan of procedure is based upon the study of the cause of death in stab wounds of the heart which I have previously discussed.[1]

The causes of death, in those cases where time is allowed for something to be done for them, are usually from one of three conditions.

(1) Heart tamponade or compression.
(2) Hemorrhage into the pleural cavity.
(3) Infection following operative procedure either in the pericardial or pleural cavities.

Upon examining the patient one can usually tell if the heart has been wounded and can also from the symptoms tell whether or not the patient is suffering from heart compression or from the loss of blood. If the thoracic cavity is filled with blood, then the wound has penetrated both the thoracic and pericardial cavities and the patient is losing blood into the thoracic cavity and may die of hemorrhage; but naturally will have the signs of hemorrhage in addition to the physical signs of hemothorax.

On the other hand, if the wound has gone through the chest wall into the pericardium without wounding the pleura, the blood will accumulate in the pericardium because it cannot escape through the stab wound. The patient therefore will have symptoms of heart compression which are entirely different from those of hemorrhage. The patient whose pleural cavity has not been wounded should be approached with great care in order not to open the pleura; while in the patient whose pleural cavity has been wounded and filled with blood, the transpleural approach is preferable. In the latter case the pleural cavity should be emptied of blood, the heart wound sutured, pericardium closed, pleural cavity closed air tight, and the air removed from the thoracic cavity and the lung immediately inflated.

To avoid the third cause of death one should never, in the hurry and excitement of the procedure, neglect the rules of asepsis, which should include a thorough débridement of the wound down to the ribs, complete removal of all blood in the thoracic cavity, closure without drainage and in every way guard against infection. I have seen two patients die of infection after the successful suturing of the heart, where if this rule had been observed these patients would probably have lived.

In conclusion I should think that if the younger staff about the hospital are trained to approach these cases with these points in mind, many more patients would be operated on early and many more of them would recover.

REFERENCE

[1] Singleton, Albert O.: Wounds of the Heart and a Discussion of the Cause of Death. Am. Jour. Surg., vol. 20, No. 3, pp. 515–532, June, 1933.

DR. ROBERT L. RHODES (Augusta, Ga.).—In 1924, I presented the report of two cases of wounds of the heart before the Southern Surgical Association, one patient alive and well—incidentally, still alive and well. In the report of the first patient I tried to stress the point Doctor Bullock has brought out. I called attention to the fact that the heart was kept beating for 45 minutes after the patient was apparently dead, by repeated massage and the use of salt solution and adrenalin. I did this to emphasize that these patients are not dead. What the phase is we do not know.

The patient who died had a wound in the left ventricle and was exsanguinated. I called attention to the fact that if I had another such case to deal with I would aspirate that blood and reinject it in the form of a ventricular infusion or into the veins.

I wish to second what Doctor Singleton just said about educating the young men to recognize and properly treat these cases. Recently we had two instances that were not recognized. Given stab wounds of the chest, the patients do not die quickly. It is not the emergency one might think. You may have time to have a roentgenologic examination made. In both of my cases the left pleural cavity was involved and there was no difficulty in recognizing the presence of a hemothorax.

I wish to add a fourth fact: these two recent cases both died of cerebral emboli. The blood clot in the ventricle was pinched off and the cerebral vessels in both cases were full of emboli. Cerebral emboli constitute, therefore, a fourth cause of death.

I would like to add one case to Doctor Shipley's report. Following the paper of Dr. George H. Bunch last year, I went home and saw a man who had had an empyema probably for three weeks before he was brought in. Three days after the empyema had been drained we found his pericardium needed to be opened, because of suppurative pericarditis. This was done and he made a satisfactory convalescence for nine days, when he wakened from his sleep about midnight and asked the nurse to hold his hand because he was going to die. She sent at once for the house officer. He could find nothing wrong, but the patient was dead within an hour. The autopsy failed to show the cause of death.

THE DEVELOPMENT OF SELECTIVE EXTRAPLEURAL THORACOPLASTY FOR PULMONARY TUBERCULOSIS

FRANK S. JOHNS, M.D.

JOHNSTON-WILLIS HOSPITAL, RICHMOND, VA.

THROUGH an era of change and progress, it is natural to anticipate variation and modification in the operative procedure of any disease; especially, of a pathology in which surgery has only recently—within the past two decades—proved its value. The early advocates of extrapleural thoracoplasty for pulmonary tuberculosis were of one opinion as to its surgical technic. A resection of the first to the tenth or eleventh ribs, inclusive, was completed in practically every case of this type. Any lesser procedure was held to be useless and thought to leave the patient in a precarious condition. So dogmatic were Sauerbruch and others on the importance of a complete surgical collapse for every case that one hesitated to change in essential detail the so called standard operation.

We should not underestimate the experience and opinion of the leaders in specific fields. Important also is the surgeon's responsibility for the significance of improved results in his own cases; that, clinically alert, he may evaluate such results in terms of surgery, and shall profit thereby. No rigid adherence to former precept, however standardized, can develop the usefulness of any surgical procedure.

For several years, in my work in the field of thoracic surgery, I performed, as generally advocated, the complete extrapleural thoracoplasty in all cases of surgical collapse. In spite of its epochal importance, the disadvantages of this extensive "complete operation" were constantly obvious. While offering hope to a large group of desperate patients, it exacted a maximum of shock, disability and economic loss, and carried a threatening operative mortality. In 1932, our first inventory of 100 cases of extrapleural thoracoplasty for pulmonary tuberculosis caused us to draw an important deduction. At that time about two-thirds of our cases had had the complete operation. The remaining 33 had had so far a *selective* operation directed simply at closing the existent cavity or cavities, with no effort made to compress any part of the lung undamaged by tuberculosis. The improvement in these cases of selective thoracoplasty was greater and more significant than the figures would indicate.

Encouraged by such pioneer results, we proceeded boldly in a more radical departure from the standard operation, finding the selective thoracoplasty suitable for an increasing number of our cases, and became convinced of its importance in the development of thoracic surgery. We had been warned of certain grave complications to be expected if we began at the top instead of at the bottom in our resections of ribs; also of what would surely follow if we did not complete the standard massive operation in every case. But

from our earliest use of selective surgical collapse, there occurred no extension of pathology into the opposite lung, nor was it followed by any extension into the healthy part of the lung under operation. The dire warnings had failed to materialize. And with serious thought, we continued to alter definitely our standard type of operation for pulmonary tuberculosis.

Having laid down as our basic principle that "one should always adapt one's operation to the pathologic condition present," we no longer routinely began at the base and resected, in stages, all the ribs. Instead, we took increasing care to *select* for every patient the type and extent of operation best suited to his individual case. If only the apex was involved, as in the majority of cases referred for surgical collapse therapy, it was carefully determined what ribs should be resected and what lengths of ribs should be removed in order to collapse the cavity or cavities completely.

Logically the next question followed: Why not exert every effort to impair as little as possible the function of the remaining healthy part of the lungs? The purpose of every extrapleural thoracoplasty of whatever degree is to close cavities and to put the diseased lung at rest. It is not directed toward limiting the function of an undamaged lung. Too often, in the collapse of any cavity, some good lung tissue is compressed. We believe, however, that the selective thoracoplasty does the least amount of damage to healthy lung tissue.

Vital capacity, as emphasized by Graham, is an essential factor in doing extensive rib resections in cases of pulmonary tuberculosis. When only the partial or selective collapse is carried out, vital capacity plays a less important rôle. If only the damaged part is compressed, the effect on the lung in this respect is frequently negligible.

In any study of a series of pulmonary collapse, it is surprising to note how few cases justify resection of all the ribs. Operable cases usually have no extensive cavitation throughout the lung. A case so unfortunate is the late neglected type rarely seen in a modern tuberculosis hospital. The phthisiotherapist, scrutinizing each case routinely and more vigorously than in the past, now promptly refers all cases suitable for surgery while their cavities are small and ready for selective collapse treatment. The neglected tuberculous patient who has been unwisely advised or treated "expectantly" too long, is the case least suitable for the operation we advocate. On the other hand, if the case has been followed from its incipiency by a capable physician schooled in tuberculosis, indications for the standard extrapleural thoracoplasty, as popularized by Sauerbruch, will be the exception. Rest, pneumothorax, pneumolysis, phrenic surgery, or finally, a selective thoracoplasty will be our usual procedure.

Bilateral cavities at the apex are readily collapsed by bilateral selective thoracoplasty. Since, by this type of case, the complete thoracoplasty could not be borne, such a patient would never be suitable for the complete operation and would be given up as hopeless. We refer with interest to the frequency of indications for apical thoracoplasty based on Medlar's recently

published pathologic theory. His experiments with sections of whole rabbit lungs have shown that tuberculosis unerringly seeks the highest area of the lung, basal or apical according to posture. He states that: "The main seat of chronic cavitating tuberculosis in rabbits and cattle is the posterior ventral surface of the posterior lobe." Due to posture, the base is thus the more elevated portion of the rabbit's lung, relative in plane to the human apex, in which location tuberculous cavities most frequently occur. It has further been proved possible to bring about a change of location of the progressive tuberculous process in the rabbit lung by changing his posture.

Our experience with selective collapse therapy dates from 1924. After five years' work we first advocated this procedure before the Association for Thoracic Surgery. No interest in our recommendation of upper or partial thoracoplasty was particularly evidenced at that time. We again ventured to report our results in 1932. When first presented before this Association in the same year, it aroused slight and conflicting discussion. At that session, Dr. Louis Franck definitely "questioned the possibility of securing the necessary and desired collapse by the plan advocated." Doctor Boland admitting that our work appealed to him, asked "If the lower portion of the lung is in good condition, why collapse it?" Still more favorably, Dr. George Bunch agreed that "with a cavity in the apex, if there is not much involvement of the lower portion of the lung, I think this type of precedure is indicated."

To date, Dolly reports 120 cases of apical thoracoplasty. O'Brien states that he too is now doing a limited thoracoplasty. Overholt, from Lahey's Clinic, reports partial thoracoplasty on 53 cases with pleasing results. Mr. W. H. C. Romais and Mr. S. H. Sellars of London report favorably on a series of such cases. I have not found any series of limited or selective surgical collapse that has shown unfavorable results. It is significant that the surgeons who have been doing this work are universally enthusiastic about it. That others are now getting satisfactory results with the selective operation, and that none of the complications formerly predicted have materialized in their cases, are matters keenly gratifying to us. It means, I frankly think, that only recently has this useful operation been fairly tried, and that little accuracy was formerly recorded as to its results. In spite of such development, however, it is apparent from a survey of today's literature that the standard operation of Sauerbruch is still the technic commonly practiced by the majority of surgeons.

The indications for a selective thoracoplasty are similar to the indications for the complete operation. We have stressed the importance of an earlier and less severe operation. But let us keep clearly in mind the fact that surgical collapse by resection of ribs should always be considered as a last resort and that no patient should be subjected to a thoracoplasty of any extent until all other suitable means of treatment have failed. There are many less severe types of collapse therapy which should be tried before we resort to rib resection; remembering, particularly in the case of resection of all the ribs, that the lung thus compressed is definitely and permanently *hors de*

combat. This warning applies also to the use of partial or selective thoracoplasty.

The selective operation differs little in technic from that of the standard thoracoplasty. Each resection must be planned according to the condition of the patient, and the size and location of the cavity or cavities to be closed. If the cavity is larger and located high in the apex, greater lengths of the upper ribs should be resected. If it is small, a relative amount of the ribs should be removed. In resections of the upper four or five ribs, we make use of an ample incision. This enables us to lift the scapula well forward so that any lengths of the first and second ribs may be exposed. An adequate exposure is vital in removing the upper ribs. We must close the cavity, regardless of the amount and number of ribs to be resected. Not infrequently, anterior sections of the ribs must be removed to accomplish a complete closure of the cavity and a negative sputum. Paralysis of the diaphragm by operation on the phrenic nerve has been popular in the surgical treatment of pulmonary tuberculosis. We have used it selectively as indicated, but not as a routine measure.

Briefly summarized, the advantages of selective thoracoplasty are these: It is less severe and less shocking; thus allowing us to operate on many patients who would otherwise be denied surgery. The number of stages of operation are fewer, since nearly all selective thoracoplasties can be done in one stage; a point important to the patient as well as to his family. Further, as above mentioned, and as our series will show, bilateral cases stand the limited or selective operation exceedingly well; this, because the healthy part of the lung continues to function without embarrassment. By virtue of such versatility, the field of thoracic surgery is distinctly broadened through the practice of a limited surgical pulmonary collapse.

The selective operation interferes very little with cough and expectoration; an important advantage. Statistics show that the standard operation of Sauerbruch predisposes to the development of pneumonia. The resection of the lower ribs interferes seriously with postoperative cough, with resulting stasis. In selective thoracoplasty, stasis is not a troublesome factor, because coughing and expectoration are little disturbed. The percentage of pneumonias following the selective operation is relatively low.

SUMMARY.—A few outstanding facts mark the survey of our series of cases of selective thoracoplasty. (1) From the present favorable condition of several patients operated on 11 years ago, the principle involved in selective collapse therapy is shown to be sound. (2) The fear of promoting an extension of the disease into healthy lung tissue has entirely failed to materialize. (3) This type of operation broadens the field of usefulness because it is less severe and more applicable to bilateral cases.

It is interesting to note that all of my cases were studied and selected through consultation with Dr. Dean B. Cole and his associates. The results here given are the reports from the medical staff. I feel very strongly that this selection of cases is the determining factor in our surgical success. Real

progress depends on early, intensive application of the type of therapy best suited to the given case.

We have done selective thoracoplasty on 67 of our cases with an operative mortality of 5.6 per cent. Since December 1, 1932, 90.4 per cent of our cases have had a selective thoracoplasty only. Our whole principle has been to close cavities, doing as little damage as possible to the healthy lung tissue; and to render the sputum free of tubercle bacilli.

THE TECHNIC OF THORACOPLASTY FOR PULMONARY TUBERCULOSIS

B. NOLAND CARTER, M.D.

CINCINNANI, OHIO

FROM THE DEPARTMENT OF SURGERY OF THE COLLEGE OF MEDICINE OF THE UNIVERSITY OF CINCINNATI AND THE CINCINNATI GENERAL HOSPITAL

TEN years ago I performed my first thoracoplasty for pulmonary tuberculosis. Since that time, 159 additional cases have been operated upon. On looking back upon this first case, which was done in 1925, it is apparent how much the present technic differs from the original one.

In 1885, De Cérenville performed what is considered to be the first real thoracoplasty. His operation consisted of the resection anteriorly of short segments of ribs over tuberculous cavities at the apex of the lung. Several other surgeons, notably Carl Spengler, Quincke and Garré performed operations quite similar to that of De Cérenville, in that very short segments of a few ribs were removed. The results of these operations were poor and the operation did not become popular. In 1907, Brauer, feeling that the poor results of these first operations were due to the removal of too short segments of a few ribs, advocated the removal in a single stage of the entire length of ribs II to IX, inclusive. Friedrich performed such an operation in 1907. Though his first patient survived, the mortality in the subsequent operations was 30 per cent. Wilms, in 1911, demonstrated clinically the value of the experimental work of Boiffin and Gourdet by the good results he obtained in cases of pulmonary tuberculosis in which he resected short lengths (3 to 5 cm.) of ribs I to VII close to the spine. The mortality was much lower than that of the Brauer-Friedrich operation. Wilms later modified his original operation in that he removed somewhat longer segments of ribs and extended the operation to include ribs I to X. Sauerbruch, working independently, advocated practically the same operation. John Alexander, in his exceptional monograph, "Surgery of Pulmonary Tuberculosis," published in 1925, speaks of this last operation as the "Wilms-Sauerbruch" and states, "It is the technique, although sometimes slightly modified, that is being used throughout the world by most surgeons of experience."

Thus it can be seen that from its beginning in 1885 up to 1925 the operation of thoracoplasty had passed through the phases of short rib resection, very long rib resection, and finally of removal of short segments of ribs. This was the technic of the operation at the time I first became interested in it. The essential features of the operation at that time were (1) the resection of 2 to 3 cm. of the first rib, 3 to 5 cm. of ribs II to VII, and 6 to 15 cm. of ribs VIII to X or XI; (2) the removal of the lowermost ribs at the first stage followed in two weeks by the removal of the upper ribs, and

(3) the resection of ribs I to X or XI in every case regardless of what was the underlying pathology in the lung.

After a few years' experience with the above described technic, it became evident to me that the operation which I was doing was not closing cavities in the lung completely, unless the cavities were small, or unless there was little fibrosis about them. This same feeling was experienced by many other surgeons and supplementary thoracoplasties were done anteriorly to increase the collapse in cases in which the original operation had proved to be inadequate. The result has been that increasingly long segments of ribs overlying cavities have been removed until at present practically the entire rib from spine to sternum is resected. This removal of great lengths of ribs over the most diseased portion of the lung is regarded as essential in the present day thoracoplasty and will, I believe, contribute more than anything else to the complete and permanent closure of a tuberculous cavity. The collapse of the chest wall as seen at operation in my early cases, with resection of short portions of ribs, was very inadequate as compared to that obtained now by the removal of practically the whole first rib and of great lengths of all ribs over a cavity. A number of my earlier cases have had to be reoperated upon in order to close cavities which were not completely collapsed because of the inadequate removal of ribs. I now feel strongly that there is no standard operation which is suitable for all patients, but that the operation has to be made to fit the needs of the individual patient, in that varying lengths of ribs and varying numbers of ribs should be removed depending on the character of the lesions in the lung.

From 1925 until 1931, I regularly resected ribs I to X inclusive in all cases and removed the lower ribs at the first stage. The rationale of the primary removal of the ribs from the lower portion of the thorax was that by compressing the lower lobes first, one prevented aspiration into them of infected material which was forced into the bronchi when the cavities at the apex were compressed. This danger of aspiration is not considered as real now as it formerly was, probably because a phrenicotomy is being done preliminary to most cases of thoracoplasty, and the amount of compression thus afforded the lower lobes guards against aspiration. It would seem more logical to remove the ribs over the upper portion of the chest first because the apex is usually the most diseased portion of the lung and contains the cavities, for the obliteration of which the operation is performed. A further reason for the primary removal of the upper ribs is that, if the second stage of the operation has to be abandoned or long delayed after the first stage, the cavity in the apex will have been completely or partially collapsed. The patient will, therefore, have a better chance of recovery than if the size of the cavity had been unchanged by an operation performed on ribs far removed from it. Therefore, for the past several years the uppermost ribs have been resected at the first stage, proceeding from above down rather than from below up, as was formerly the case.

I find myself growing more and more conservative in the amount of

operating done at any one stage. It has been found to be wiser to err on the side of doing a good deal less than the amount the patient will tolerate rather than doing too much. Whereas formerly five ribs, and sometimes six, were resected at a single stage, at present only three ribs and occasionally

FIG. 1.—The auscultatory triangle.

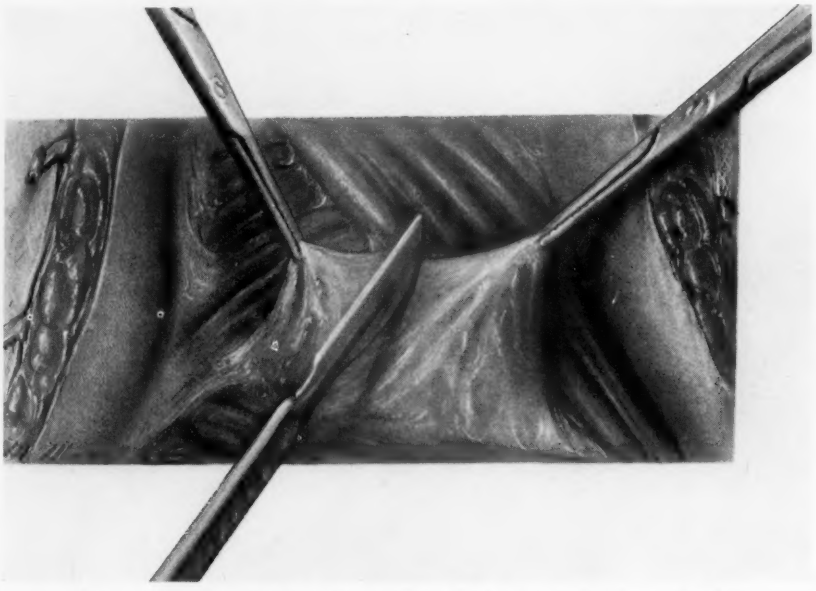


FIG. 2.—The division of the loose areolar tissue in the auscultatory triangle.

four are removed at one sitting. One can never predict just how much a tuberculous patient can tolerate, and it is a much wiser policy to stay well within the limit of safety in each of three stages of an operation than to be

on questionably dangerous ground in each of two stages. When the operation is broken up into many stages, or when it is thought that more than two weeks may elapse between operation, 10 per cent formalin solution or

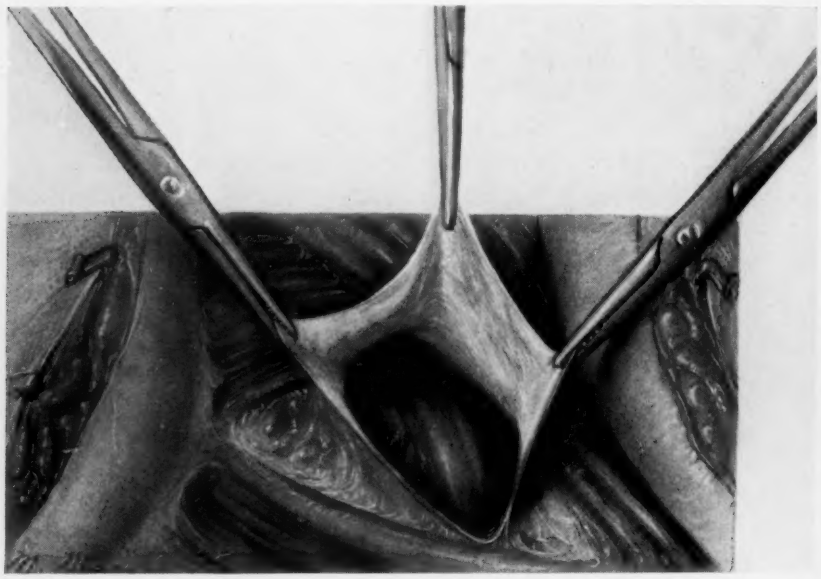


FIG. 3.—The areolar tissue in the auscultatory triangle has been divided and the cleavage plane between the sacrospinalis and overlying muscles exposed.

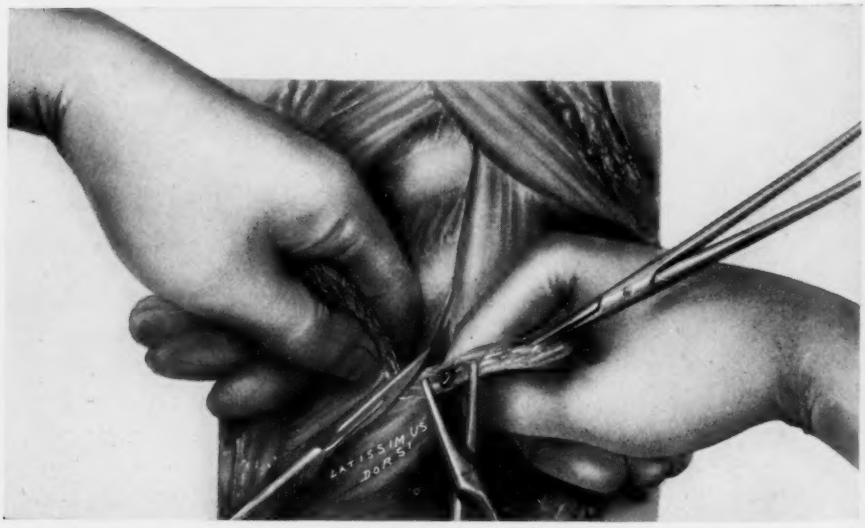


FIG. 4.—The latissimus dorsi muscle is being divided. Note the "tourniquet" effect of the thumb and forefinger on the left. On the right the muscle has been slightly released and a bleeding point thus rendered visible is being grasped with a hemostat.

Zenker's solution should be applied to the periosteum of the resected ribs. This prevents the regeneration of the ribs until the ultimate degree of collapse has been obtained.

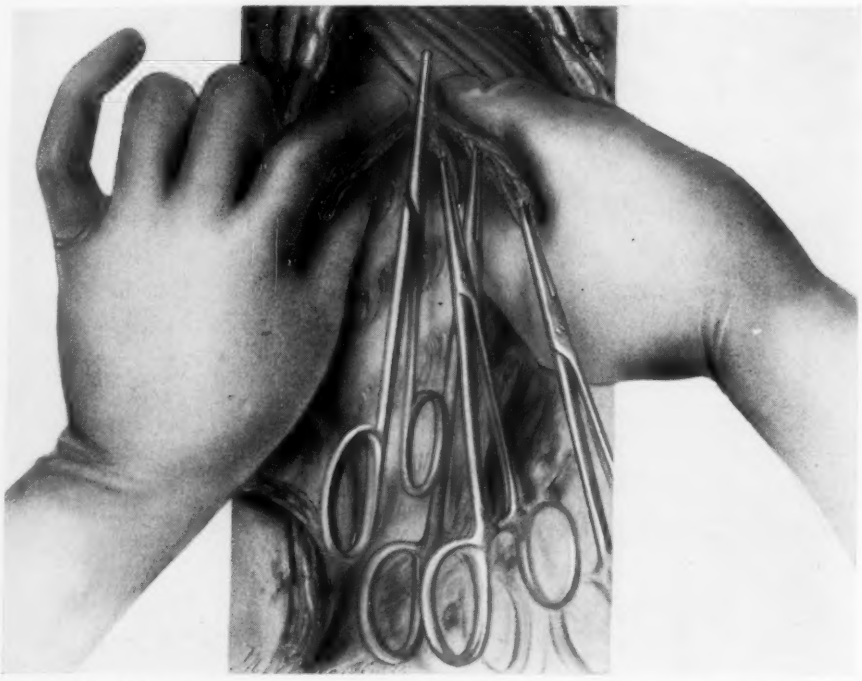


FIG. 5.—The division of the trapezius and rhomboids. The same maneuvers as in Fig. 4 are being carried out.

FIG. 6.—The separation of the trapezius muscle from the underlying rhomboid muscles.

It should always be borne in mind that the patients upon whom thoracoplasties are performed are potentially or actually poor risks and may easily be thrown into shock by loss of blood or by unnecessary traumatism to their tissues. In our clinic meticulous and gentle handling of tissues is insisted upon. Sharp dissection is done in contrast to the pulling and tearing of tissues; vessels are isolated where possible and divided between hemostats; cleavage planes are closely followed. Halsted hemostats are used exclusively, silk sutures and ligatures are used throughout the operation, and the wound is closed without drainage with interrupted sutures. All of our operations are performed under local anesthesia which, while more time-consuming and perhaps more trying on patient and surgeon, certainly tends toward more careful and gentle handling of tissues.

FIG. 7.—The trapezius muscle has been separated from the rhomboids and is being divided. The underlying rhomboid muscles will be treated in the same manner.

A thoracoplasty is an operation of considerable magnitude, involving a very long incision with a wide exposure of raw surface. During its performance it is easy to lose more blood than one realizes, and loss of blood is a serious matter to the tuberculous patient who frequently has an anemia of some degree and in whom the general resistive powers are none too good. I make every effort to prevent loss of blood. The operation is carried out only as rapidly as is compatible with accurate hemostasis. All bleeding points are clamped, the larger vessels are ligated, the smaller ones are controlled with the Bovie unit. The steps in the operation which are necessary for an adequate exposure of the ribs and which carry with them the possibility of the loss of considerable blood are (a) the division of the heavy muscles of the back, *viz.*, the trapezius, the latissimus dorsi, and the rhomboids; (b) the

mobilization and retraction of the sacrospinalis muscle, and (c) the division of the serratus anterior muscle.

Two years ago I described a method by which the back muscles could be divided with little loss of blood. This consisted in dividing the loose areolar

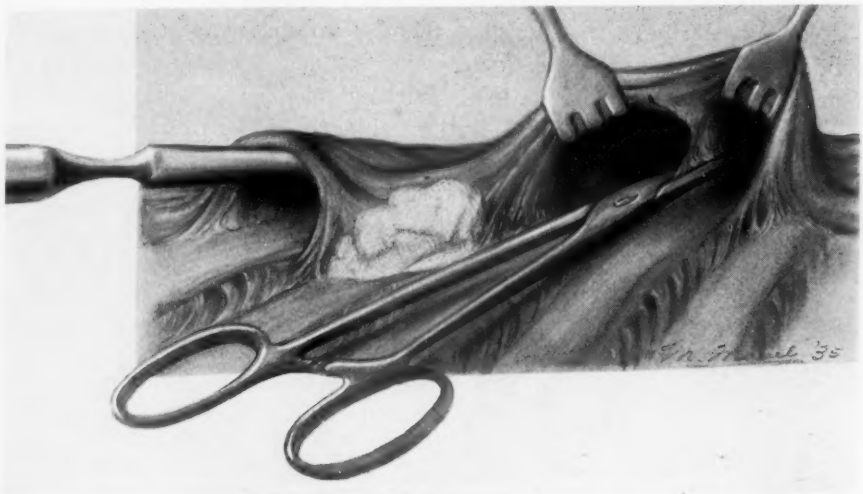


FIG. 8.—The mobilization of the sacrospinalis muscle.

FIG. 9.—The sacrospinalis muscle has been mobilized and retracted exposing the articulation of the rib with the transverse process.

tissue in the auscultatory triangle, in finding the cleavage plane between the sacrospinalis muscle and the overlying trapezius, rhomboids and latissimus, in freeing the latter muscles and in grasping them between thumb and finger

before dividing them. (Figs. 1, 2, 3, 4 and 5). By a slight release of pressure on the muscle the bleeding points can be seen and caught with hemostats before the vessels retract into the muscle. This method is still found to be satisfactory. It has been modified in that at the point where the trapezius muscle becomes quite thick, which is about opposite the fourth spinous process, it is separated from the underlying rhomboid muscle, then each muscle is divided individually, using the "tourniquet" method. (Figs. 6 and 7). This simplifies the procedure for when the muscles are thick it is difficult to compress them thoroughly. There is a large branch of the transverse cervical

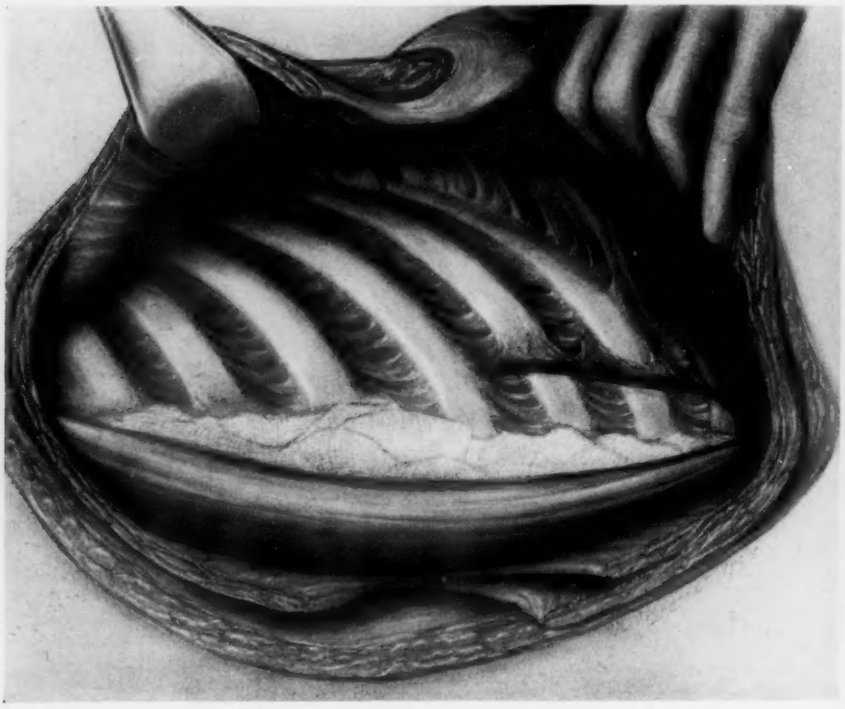


FIG. 10.—The scapula has been lifted, exposing the serratus anterior muscle.

artery which courses in the plane between these two muscles and is rather elusive unless it can be seen before it is divided. It is important to divide at one time only as much muscle as can be grasped, and to completely control the bleeding from its cut edge before proceeding with the grasping and division of the remainder of the muscle. The smaller vessels along the cut edges of the muscle are coagulated with the Bovie unit, but the larger ones are transfixed with fine silk.

The resection of the ribs close to the spine has always been essential in a properly performed thoracoplasty. However, within the past two years there has been a tendency to go farther still and to resect the transverse processes of the vertebrae also. The complete mobilization of the sacrospinalis muscle

is necessary in order to expose the articulations of the ribs with the transverse processes. (Figs. 8 and 9). A short incision is made at the outer edge of the muscle at the point where it crosses the rib. A narrow periosteal elevator is passed between the rib and the overlying muscle, hugging the rib and following it back to junction with the spine. This process is repeated on each of the ribs which are to be resected and on one or two ribs above and below them. Small retractors are hooked under the edge of the muscle where it has been freed from the ribs and the muscle is elevated. It will be found that the complete mobilization of the muscle is prevented by some fleshy

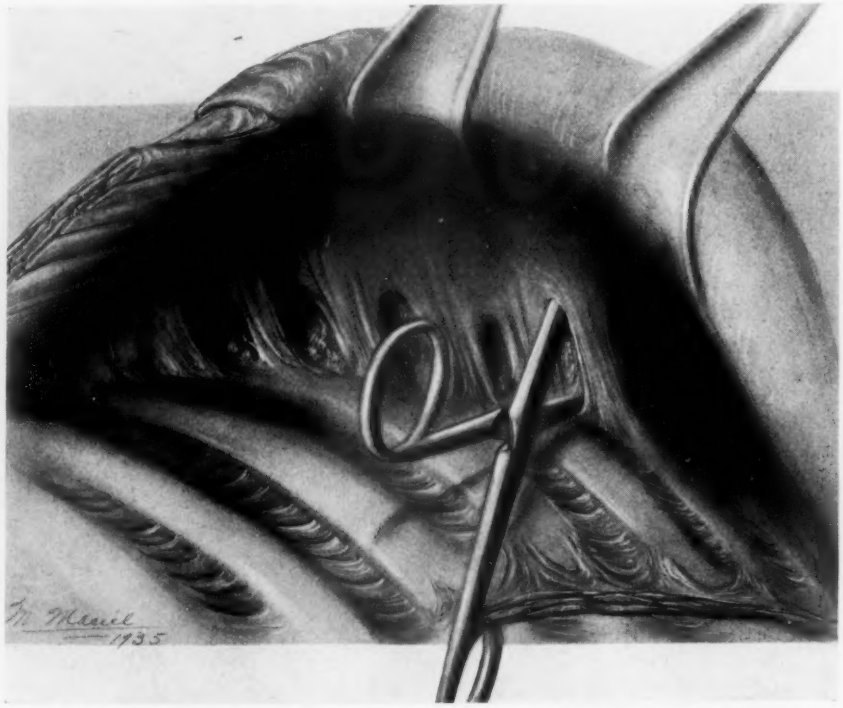


FIG. 11.—The serratus anterior muscle has been split up into several small portions.

fibers which attach it to the intercostal space. In each interspace there is a small artery which runs in these fibers and which supplies the sacrospinalis muscle. If these intercostal attachments of the sacrospinalis muscle are grasped with a hemostat before being divided, there is little loss of blood. After these intercostal attachments have been cut, it will be found that the muscle can be retracted medially so as to expose the transverse processes.

Experience has shown that it is essential to resect very long segments of the ribs overlying a cavity—this is in marked contrast to the procedure in the earlier thoracoplasties in which much shorter segments of the ribs were removed. Since practically every cavity is in the upper portion of the thorax, one is confronted with the necessity of gaining exposure of the entire lengths

of the upper ribs. When one attempts such an exposure he is confronted with the serratus anterior muscle which is attached to the upper six ribs along the posterior axillary line (Fig. 10) and, unless this muscle is divided, the portions of the ribs anterior to it cannot be reached. This muscle varies in size, but as a rule is at least one-half to one inch in thickness. Running in its substance are several large vessels. In addition, the axilla with all its structures lies just anterior to it. The problems in the division of the ser-

FIG. 12.—The lower portions of the serratus anterior muscle have been divided. The axillary contents are being pushed forward by the gauze sponge.

FIG. 13.—The serratus anterior muscle has been completely divided. Note the axillary vein, artery and brachial plexus as seen from the back, also the additional exposure of the ribs beyond the cut edge of the serratus muscle.

ratus anterior muscle are then to be able to control the bleeders in its substance, and not to injure the axillary contents which lie very close to it. These problems are met as follows: The scapula is elevated by deep retractors or an assistant's hand so as to put the serratus muscle on a stretch; starting below, at the level of the fifth or sixth rib, the muscle is split in the direction of its fibers with long handled scissors, just as one does the internal oblique muscle in making a McBurney incision (Fig. 11); other such splits are made

parallel to the first about one inch apart all the way up to the first rib. Thus the large muscle is divided up into many small bundles each of which can be divided and any vessel in it readily grasped with a hemostat. The lowermost bundle is divided first and the lower axilla entered from behind; a curved sponge stick grasping a small gauze sponge is now introduced through the opening in the muscle and the axillary contents gently pushed forward away

FIG. 14.—The injection of the intercostal spaces with ½ per cent novocaine. Note that the injection is made close to the spine, the interspace being palpated through the sacrospinalis muscle.

from the muscle (Fig. 12). The individual muscle bundles are now divided with scissors. The densest attachments of the serratus anterior are to the first and second ribs and when these have been divided the scapula can be lifted far off the chest wall. After complete division of the serratus anterior muscle (Fig. 13) the axilla is clearly seen from the back, the axillary vein and artery and the brachial plexus are all identified, and the ribs can be followed around to their cartilages if desired.

As stated previously, all of our operations except a very few have been done under local anesthesia. Direct infiltration of the skin, subcutaneous tissue and muscles with ¼ per cent novocaine is done before the incision is made. When these structures have been divided and the intercostal spaces exposed, each space is injected with ½ per cent novocaine close to the spine by palpating it through the sacrospinalis muscle (Fig. 14). The anesthesia is very satisfactory in practically all cases and it is most unusual for the patients to complain of pain, though sometimes the retraction of the scapula gives discomfort as does the freeing of the first rib. The safety of having the patient's cough reflexes present and the fact of not being fearful of a long inhalation anesthesia more than outweigh the somewhat greater tediousness of the operation under local anesthesia.

The wound is carefully inspected before closure to be sure that all bleeding points have been controlled. The muscles are closed accurately with interrupted sutures of silk, as is the skin. None of the cases has been drained. Subsequent stages are done at two week intervals or as nearly so as possible.

DISCUSSION OF THE PAPERS OF DOCTORS JOHNS AND CARTER

DR. FRANK H. LAHEY (Boston, Mass.).—I do not wish to pose as a thoracic surgeon. I say this not boastfully but regretfully, because this work is so stimulating. I think that any group that has had any experience with chest surgery should take occasion to present their conclusions and results, for in this field, which is making so much progress, it is necessary to present figures which will be interesting and stimulating to everyone involved.

As to bronchiectasis, Doctor Overholt, who does the chest surgery in our Clinic, has done eight lobectomies. He feels that one should make no decision as to one or two stage procedures before deciding as to whether or not there are adhesions between the pleura and the lobe, but when there are adhesions the operation may be done in one stage. When done in two stages, it is of great advantage, he feels, first for the chest surgeon to cut the inferior pulmonary ligament which permits the lower lobe to contract and then the upper lobe to expand and fill the cavity. He has performed lobectomy in eight cases with no operative deaths and all in good health at present. Some are not completely free of cough and expectoration, but they are greatly improved. Five were done in one stage and three in two stages. He has, at the present time, completed a two stage lobectomy on the right side in one individual and the first stage of a left lower lobectomy in the same individual.

Everyone who is performing thoracoplasty is indebted to Doctors Cole and Johns for establishing the use of the procedure from above downward. Just as in subtotal thyroidectomies, these patients need time to recover from the first stage of the operation. They need a time to be up and around. If you make roentgenograms within two weeks the lungs are still cloudy. If you do the operation at the end of two months, the patients are strong and in good condition, and it is easier to perform the secondary procedure. Our chest surgeons strongly believe that the period of time between stages of the thoracoplasty operation should be somewhere between four and eight weeks. There must be an optimum time and it cannot be within a period of ten days to two weeks. As a general surgical principle, we do not feel that patients should be subjected to a second major operation until after they have obtained all possible benefits from the first procedure. In view of the fact that rapid

successive stage operations in thoracoplasty work is preached, and carried out, in so many places in this country, it is the feeling of our men that it is very important to emphasize the necessity of this delay in the second stage operation.

Our chest service is accepting bilateral cases for thoracoplasty if there is at least one functioning lobe on each side, and if the vital capacity is not under 1,100 cc. Doctor Overholt and his associates have done 575 thoracoplasties in 250 patients in the Clinic with an operative mortality of 2 per cent and a patient mortality of 4 per cent. In a recent analysis of the first 100 patients operated upon, the following results have been obtained: Improved, 88 per cent; unimproved, 1 per cent; operative deaths, 4; sanatorium deaths, 7; apparently arrested cases, 66. (Of these 36 have been discharged from the sanatorium.) Approximately 20 bilateral cases were included in this first series of 100 patients.

As to primary carcinoma of the lung, here I think is another exceedingly stimulating field. Eighty per cent of the cases upon whom Doctor Overholt has operated had lesions in the stem bronchus which were visible through the bronchoscope. There is today an increasing tendency to make roentgenograms of patients, with obscure chest symptoms, and interpretation of chest films has improved. Bronchoscopy is being carried out more frequently than formerly. The value of diagnostic pneumothorax and intrapleural thoracoscopic examination is recognized, and the use of aspiration biopsy will assist in the diagnosis of obscure peripheral lung tumors. When one realizes that there is a tendency for these primary growths of the bronchus to be relatively benign, and that the carcinoma is surrounded by cartilage which tends to protect it from extension, this becomes a relatively hopeful field.

We are more and more impressed with the value of cyclopropane, and we believe that all intrathoracic operations should be carried out under intratracheal anesthesia, preferably with this agent. We routinely use oxygen therapy for the first two or three days following operation.

Doctor Overholt has carried out two lobectomies for malignancy of the lung. Both patients recovered; one lived ten months and died of mediastinal metastases. The other patient, who had a right upper and middle lobe resection, is living at present without evidence of metastasis. He has carried out six total pneumonectomies for malignant disease, with three operative recoveries, and all of these patients are living, one 25 months, one 19 months, and another nine months, all without evidence of recurrence. In addition to the six pneumonectomies for malignant disease, he has done two successful total pneumonectomies for suppurative disease of the lung. We have recently received letters from 12 men in this country who have been doing most of the work of this kind and have summarized their results together with those cases reported in the literature, and find that up to 1933 four pneumonectomies for malignancy of the lung were attempted—all operative deaths. We find that since 1933 there have been 34 pneumonectomies attempted and completed with operative recoveries in 18 and of these 18 ten patients are still living. It is also interesting to report that 12 of these 34 pneumonectomy cases were done in Boston, six by Doctor Churchill and six by Doctor Overholt. Doctor Churchill had four recoveries and we have had three operative recoveries. However, two of Doctor Churchill's patients have since died of metastases so that there are five of the ten living pneumonectomy cases done in Boston, two by Doctor Churchill and three by Doctor Overholt.

When we realize that previous to the establishment of the Department of Chest Surgery in our Clinic we had made the diagnosis once of primary carcinoma of the lung and that in the past four years we have made the

diagnosis 33 times, it becomes evident how many cases are being overlooked. The outstanding feature has been intractable cough. When one realizes that it produces atelectasis early, that this is recognizable by roentgenologic examination, and that 80 per cent can be recognized by the bronchoscope, one realizes that this is a field which offers really great promise.

DR. W. P. HERBERT (Asheville, N. C.) (guest).—We have found in a number of cases of arrested tuberculosis that there is what one might call a bronchiectatic state existing in them, in either one or both lungs. Not a true bronchiectasis but one which causes considerable discomfort to the patient and is the source of some irritation. Two years ago I reported two cases operated upon for this condition. Since that time there have been two more. The operation as described consisted in removing the rib above the lesion thus making a gutter so that dense adhesions could form with the pleura. In this way one gets more or less fixation of the lung. Following this a resection of the ribs over the bronchiectatic area is performed. This in turn is followed by a phrenicectomy. Thus a collapse of that portion of the lung which is causing the trouble is effected. As I have said before, I do not advocate this in a massive bronchiectasis, but I feel that in some instances this procedure is of value.

DR. FRANK S. JOHNS (closing).—We have not felt that it was necessary or wise to remove the entire upper third and fourth ribs in all of our cases. If we have a large cavity and we think it is essential to remove the entire rib or ribs to close this cavity, we do so. For smaller cavities we remove shorter lengths of the ribs.

Doctor Lahey is correct in emphasizing the importance of ample time between the stages of selective collapse for pulmonary tuberculosis. These cases should be given an opportunity to get the full benefit of the previous stage of the operation.

THE SURGICAL TREATMENT OF CONGENITAL TRACHEO-ESOPHAGEAL FISTULA IN THE NEW-BORN

MIMS GAGE, M.D., AND ALTON OCHSNER, M.D.
NEW ORLEANS, LA.

FROM THE DEPARTMENTS OF SURGERY, TULANE UNIVERSITY SCHOOL OF MEDICINE AND TOURO INFIRMARY, NEW ORLEANS, LA.

CONGENITAL malformations of the esophagus are of various types, but fortunately these anomalies are not common. They occur, however, with a greater frequency than is commonly thought, as evidenced by our experience, having seen six cases during 15 months. Brenneman[1] reports the observation of five cases in five years. We believe that as one becomes familiar with the clinical manifestations of these lesions, the incidence will increase. There is no doubt that a large number of cases that supposedly die of birth injuries, especially in those with cyanosis, atelectasis, or postdelivery pneumonia, are cases of undiagnosed congenital tracheo-esophageal fistula.

Although tracheo-esophageal fistulae are the most frequent congenital anomalies of the esophagus, there are many other embryologic defects. Strong and Cummings[2] suggest the following classification of esophageal anomalies:

(A) Involvement of the entire passage
  (1) Stenosis
  (2) Total atresia (reduced throughout to a solid cord)
  (3) Agenesia (no detectable vestige of the structure)
  (4) Doubling of the whole length of the passage.
(B) Regional involvements
  (1) Stenosis (single or multiple stenotic intervals)
  (2) Atresia (either by the narrowing of an interval or by the presence of a barrier fold, or membrane in an esophagus otherwise normal)
  (3) Agenesia (no detectable remnant of a restricted interval)
  (4) Fistula communicating with trachea or a primary bronchus
    (*a*) Esophagus otherwise normal
    (*b*) Esophagus divided into upper and lower segment by a regional stenosis, atresia or agenesia, the fistula being usually single and uniting the lower segment with the trachea
  (5) Doubling in a short extent
  (6) Diverticula
  (7) Cysts

The Group B.4b anomalies are the most frequently encountered. It is these that will be considered in the present discussion. The lesion consists of stenosis, atresia, or agenesia of the esophagus, usually the formation of

a blind pouch at the distal end of its proximal segment and a fistula between the trachea and the distal esophageal segment.

According to Mackenzie,[3] the first recorded case was by Durston, in 1670. He reviewed 62 cases of malformations of the esophagus, of which group 40 (66.1 per cent) had atresia, with the lower end of the esophagus joining the trachea just proximal to the bifurcation. Plass,[4] in 1919, reported a review of 136 collected cases of esophageal atresia with tracheo-esophageal fistulae. In 1926, Hacker and Lotheissen[5] reported 223 cases of malformation of the esophagus, of which 173 (77 per cent) had a communication between the lower segment of the esophagus and the trachea. In 1931, Rosenthal[6] reported 255 cases of congenital esophageal atresia. In 215 (84.4 per cent) there were tracheo-esophageal fistulae. From the above statistics it is evident that the most frequent congenital malformation of the esophagus is an atresia with a tracheo-esophageal fistula. The most common type is that in which there is a segmental agenesia resulting in the formation of a blind esophageal pouch at the distal end of the upper segment and a fistula between the lower end of the trachea and the proximal end of the distal segment of the esophagus which is otherwise normal.

The genesis of this unusual anomaly is explained by a number of theories, some of which are fanciful, while others certainly are based on embryologic dysfunctions that are responsible in part or in whole for the formation of tracheo-esophageal fistulae.

The esophagus and trachea are developed from the primitive foregut. In the young embryo (3 Mm. to 4 Mm.) they are one tube which subsequently becomes divided by the tracheo-esophageal septum derived from a longitudinal projection from each lateral wall, which meet in the midportion of the primitive foregut (Fig. 1). Early in the development the trachea is only partially separated from the esophagus. The anomalous development of this septum that separates the trachea from the esophagus, forming two distinct tubes, is responsible for tracheo-esophageal fistulae. The lung bud anlage is the ventral outgrowth from the primitive foregut at the site of the future tracheal bifurcation, disturbances in the development of which may account for incomplete septal closures in the distal portion.

Luschka[7] and Klebs[8] believe that intra-uterine fetal infection or trauma are responsible for these defects. It has been suggested that normally in the development of the esophagus its lumen is patent up to approximately five weeks. Following this, however, the esophageal lumen becomes obliterated as the result of excessive epithelial proliferation, the lumen subsequently being reestablished by a process of vacuolization. Kreuter[9] suggests esophageal atresia results when vacuolization does not occur. Schridde,[10] however, in the study of 50 human embryos, found a normal lumen in the esophagus at all stages. Forssner[11] demonstrated that the separation of the trachea from the esophagus occurred at an early stage (4 Mm. to 5 Mm. embryo), whereas the epithelial obliteration of the lumen of the esophagus does not occur until the embryo is 20 Mm. in length. Streeter[12] and Rosen-

thal[6] suggest that there is a great similarity between the two developmental anomalies, spina bifida and tracheo-esophageal fistula. The latter author states in his conclusions: "The development of the anomaly seems to rest on an early fundamental change in the entodermal cells that are to give rise to the esophagus, and not on primary concomitant abnormalities. This change may be genetic and related to the anterior end of the neurenteric canal." Strong and Cummings[2] state that "the agencies predisposing to the anomaly are to be sought either in the reproductive cells from which the child was produced or in an unfavorable intra-uterine environment obtaining during the critical early period of embryonic life." They believe it is entirely a growth deficiency occurring in the two lateral intralumenal entodermal projections, the anlage of the tracheo-esophageal septum. According to Lateiner,[13] there is in addition to the lateral mesenchymal ridges which divide

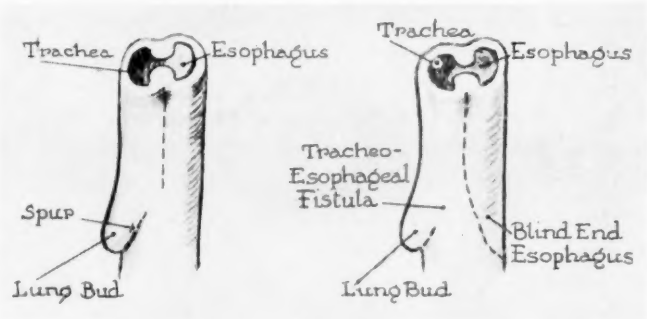


FIG. 1.—Drawing showing the spur formation of the lung bud which normally meets with the two mesenchymal ridges, separating esophagus from trachea. It is failure of this spur to meet the two lateral mesenchymal ridges which causes the spur to attach itself to the posterior wall of the esophagus, thus creating an esophageal atresia and agenesia with tracheo-esophageal fistula. (After Lateiner[13].)

the foregut into the trachea and esophagus a spur formation from the lung bud anlage, which spur grows upward into the foregut, meeting the two lateral ridges. He believes that when there is failure of the spur to join the lateral ridges and a fusion of the septum with the posterior wall of the foregut, the typical lesion results; *i.e.*, atresia of the proximal esophageal segment and fistulous communication between the trachea and the distal esophageal segment (Fig. 1). This abnormality is also dependent upon growth deficiency and the entire abnormal process may be expressed by Rienhoff's[14] law: "There is in the life cycle of a cell or tissue, which is differentiating or growing, a period in which the cell is very sensitive to its environment and any injury of it at this stage will result in the abnormal growth and development of that cell or structure." We ascribe to the theory of Lateiner[13] as being the most plausible. For a more detailed description of the different theories, the reader is referred to the excellent papers of Plass,[4] Hacker and Lotheissen,[5] Rosenthal,[6] and Strong and Cummings.[2]

The gross pathologic picture is remarkably constant in cases seen at

autopsy. In over 80 per cent of the cases there is an atresia with agenesia of the esophagus, dividing the esophagus into two distinct parts with an upper segment which terminates in a blind pouch about 10 to 12 cm. from the alveolar region. The pharyngeal part of the upper segment is normal. The end of the proximal segment is on the average 3.4 cm. below the larynx (Plass[4]). The distal esophageal segment, as a rule, is normal except at its proximal end where it is narrow and forms a fistulous communication with the trachea and rarely with one of the main primary bronchi (usually the right). The average distance of the fistulous opening above the bifurcation of the trachea is 0.5 cm. (Plass[4] and Rosenthal[6]). In most cases the upper blind pouch is entirely separated from the lower segment (agenesia), but occasionally there may be a fibrous cord or muscle band connecting the distal limb of the upper segment to the proximal end of the lower segment (Fig. 5). The tracheo-esophageal fistula is situated just proximal to the bifurcation and consists of a small, slitlike opening, resembling the mouth of a small bronchus (Fig. 8). Bronchoscopically, the fistula appears as the opening of a bronchus. The fistula is so formed that above it the posterior wall of the trachea is depressed, forming a gutter extending down to the fistula, the lower edge of which extends into the lumen of the trachea, producing a valve trap. This results in deviation of secretions and fluids from the trachea into the fistula.

The lungs reveal varying degrees of pneumonia and atelectasis due invariably to the aspiration of fluids and food into the tracheobronchial tree from an esophageal overflow. There is a high incidence of concomitant congenital abnormalities of other organs (Kelly,[15] 60 per cent; Hacker and Lotheissen,[5] 38 per cent; and Plass,[4] 62 per cent). The most common associated anomalies are (1) those in the lower rectum and anus, atresia ani being the most common (Plass,[4] 25.5 per cent), and (2) those in the genitourinary tract; *i.e.,* horse-shoe kidney, unilateral kidney, double and single ureters with and without stricture. Atresia duodeni, harelip, and other anomalies are rare. However, the majority of associated anomalies are not incompatible with life, and, therefore, they in themselves are no contra-indication to the treatment of tracheo-esophageal fistula.

The symptomatology of esophageal atresia and tracheo-esophageal fistula is so constant that the lesion should be easily recognized. The symptoms can be divided into two main groups: (1) gastro-intestinal, and (2) pulmonary. The infant at birth is well developed and appears normal except for an excessive amount of mucus in the mouth, which is probably due to its accumulation in the blind esophageal pouch and emptying into the pharynx. At times the secretion is so abundant that it interferes with breathing and attacks of cyanosis occur. These symptoms result from filling and emptying of the blind pouch of the proximal segment of the esophagus. The abdomen rapidly becomes distended by the ingress of large quantities of air from the trachea through the fistula into the stomach and small intestine. This is determined both clinically and by roentgenograms (Figs. 3, and 7). The most classic

symptoms are referable to the pulmonary system and occur dramatically at each feeding. At the first attempts at feeding, nothing happens for a few seconds, but soon a definite train of events occur. There is a cough, fluid which is often frothy regurgitates through mouth and nose, and early cyanosis occurs, and more fluid is projected through the nose and mouth as the blind pouch of the esophagus and bronchial tree is emptied of its fluid and food. Almost lifeless relaxation occurs, the cyanosis disappears, and the infant recovers to go through the identical process at the next feeding. It is simply a picture of pulmonary aspiration of fluids which may produce drowning at the first feeding, or, as more often happens, in a few days the infant succumbs to an aspiration pneumonia. These symptoms are due to emptying by reverse peristalsis of the contents of the filled blind esophageal pouch into the hypopharynx and the subsequent spilling into the trachea, some of the fluid passing into the lower segment, then into the stomach by way of the tracheo-esophageal fistula. However, most of the fluid enters the tracheobronchial tree, resulting in suffocation and occasionally death. There naturally follow rapid loss in weight and dehydration due to inability to take fluids and nourishment.

The diagnosis is not difficult if the condition is only considered. However, the majority of cases are never diagnosed until the typical symptoms appear at the first feeding, which is undesirable because the hopelessness of these cases in the past has been due to the early onset of aspiration pneumonia. We believe that the diagnosis should be made at birth. A catheter should be passed gently into the esophagus routinely in all new-borns. In this way a diagnosis can be made before there is any aspiration of the mouth secretions. If a new-born has an excessive flow of mucus from the mouth and the abdomen becomes rapidly distended (a valuable diagnostic sign, according to Brennemann,[1]) the upper esophagus should be immediately investigated. In such cases, we suggest that a catheter be passed into the esophagus and if an obstruction is met one can almost with certainty diagnose the presence of an esophageal atresia. If the catheter meets an obstruction from 10 to 12 cm. from the alveolar ridge then lipiodol should be injected into the esophagus and a roentgenogram taken. This should include the thorax and abdomen. If esophageal atresia and tracheo-esophageal fistula are present, the blind pouch is outlined by the lipiodol. There is usually some spilling of lipiodol into the trachea and lung; and the stomach and small intestine will be filled with gas (Fig. 3). We cannot overemphasize the necessity of using lipiodol instead of barium, as the latter is contraindicated due to its irritating action on the lung parenchyma. If this routine as suggested is followed, then, and not until then, will the surgeon secure the infant free of pulmonary complications.

The treatment of this unfortunate anomaly is entirely surgical. The ideal operation would be the separation of the esophagus from the trachea and an end-to-end anastomosis of the upper to the lower segment. Such an extensive intrathoracic procedure is not justified in a new-born infant, however, and the operation would always be finished as a postmortem procedure. There are two definite surgical indications: (1) closure of the fistula, and

(2) an avenue (gastrostomy) through which the infant can be nourished. Richter,[16] in 1913, suggested and performed the first intrathoracic ligation of the proximal end of the distal segment of the esophagus to close the fistula. He reported two cases that terminated unsuccessfully. We believe that this transpleural ligation is too formidable for an infant two to three days old, and have not used the procedure. We believe that the simplest procedure should be used. Therefore, we recommend intra-abdominal ligation of the cardiac end of the esophagus with umbilical tape to prevent regurgitation into the lung of gastric contents through the tracheo-esophageal fistula, and the formation of a gastrostomy of the Ssabanejew-Frank[17] type.

The technic of the operative procedure is as follows: Through an upper left rectus paramedian incision the peritoneum is opened and the stomach

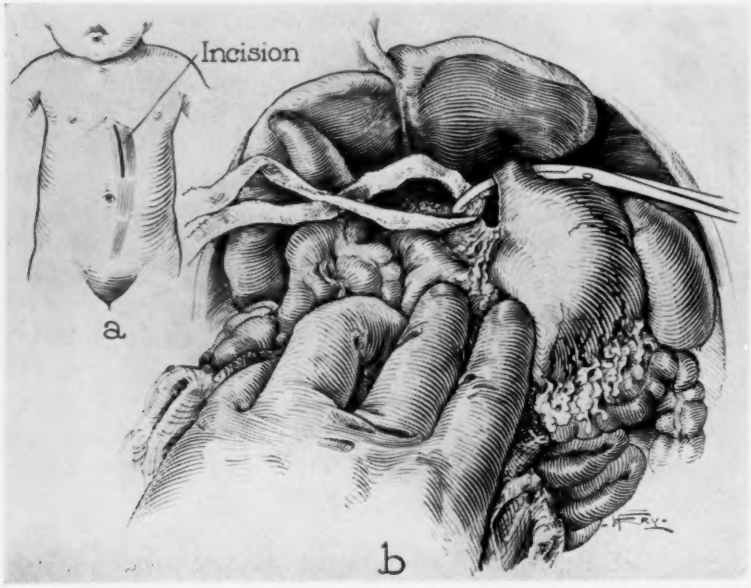


FIG. 2.—Drawing demonstrating the left rectus incision and the method of adjusting the umbilical tape ligature around the cardiac end of the esophagus.

grasped gently by "gauze covered fingers" of the left hand and retracted downward. The left lobe of the liver is elevated upward out of the field by a moist gauze covered retractor. The stomach is then depressed downward and backward. This brings the cardiac end of the esophagus to view just beneath the diaphragm (Fig. 2). A right handed aneurysm needle threaded with catgut is gently passed around the esophagus, the catgut grasped and tied to a ligature of umbilical tape. The needle is left *in situ*; the catgut is removed from right to left, pulling the tape behind the esophagus. This tape ligature is gently but firmly tied, which obliterates the lumen of the esophagus. A curved forcep can be used to pass behind the esophagus and pull the ligature of tape around the esophagus (Fig. 2). However, we recommend the aneurysm needle, as the procedure can be accomplished with

greater rapidity. The vagi are incorporated in this ligature. This cannot be avoided, but we feel that there will result slight if any deviation from the normal physiologic activity of the gastro-intestinal tract.

A gastrostomy is now formed according to the technic of Ssabanejew-Frank as follows: The stomach is grasped with Allis forceps near the greater curvature close to the cardia and by pulling on the Allis forceps a pouch of the anterior gastric wall is formed. This pouch is now pulled under, or over the rectus muscle through a tunnel in the tissues, making its exit through a secondary incision about 3 cm. from the left rectus incision. The pouch is attached to the skin of the secondary incision and the left rectus incision is closed with silk. The tip of the pouch is now cut off and a catheter can be passed easily into the stomach. The advantages of this form of gastrostomy are many: the procedure is rapidly performed, there is a tube lined with epithelium which prevents stricture of opening, and with pressure over the tunnel through which the pouch passes spilling of gastric contents between feedings does not occur. We believe these two procedures are the simplest that can be performed and yet accomplish the desired results, and both should not take over 20 to 25 minutes in their execution.

The postoperative treatment consists of administering fluids and food by the gastrostomy. Fluids should also be given by hypodermoclysis. The upper blind pouch of the esophagus should be aspirated frequently to prevent overflow into the trachea. If this simple procedure is not done aspiration pneumonia may result. The lower blind pouch created by the operation will probably take care of itself for a while, but will ultimately have to be treated by the intratracheal route. We believe that as soon as possible the upper segment should be removed to the outside of the neck as a preliminary to a future plastic operation to form an anterior thoracic esophagus. The lower segment as well as the fistula opening must be obliterated to prevent infection, because this infected material will be aspirated up into the tracheobronchial tree, producing a pneumonitis.

For a successful outcome in these cases the emergency treatment as well as the future formation of a new esophagus depends entirely upon the cooperation of the obstetricians, pediatricians, surgeons, thoracic surgeons, and the bronchoscopists. It is by the combined efforts of the above named specialists that the present mortality of 100 per cent should decline rapidly to a lower level and a few lives may be saved.

CASE REPORTS

Case 1.—Premature white male, born October 22, 1935, at 1:45 A.M. The infant cried immediately after delivery. However, respirations soon became difficult and cyanosis occurred. Cyanosis and difficult respirations were promptly relieved by aspirating mucus from the hypopharynx. These attacks recurred at frequent intervals and were relieved by removal of the excessive mucous secretions. The infant was put to the breast every two hours beginning at 10:00 A.M. the date of birth. It was noted that all attempts at nursing resulted in regurgitation of food through both the mouth and the nose. The infant struggled for air, cyanosis rapidly appeared, and it seemed that death would supervene. However, the

infant finally started breathing normally and the cyanosis disappeared. Due to the repeated attacks of cyanosis the infant was put in the Drinker respirator.

A catheter was passed into the esophagus where it met with an obstruction about 12 cm. from the alveolar margin. Lipiodol was introduced into the upper end of the esophagus and a roentgenogram was made. This revealed a blind esophageal pouch of the upper segment of the esophagus. The stomach and the small intestine were filled with gas (Fig. 3). A diagnosis of esophageal atresia with agenesia and tracheo-esophageal fistula was made.

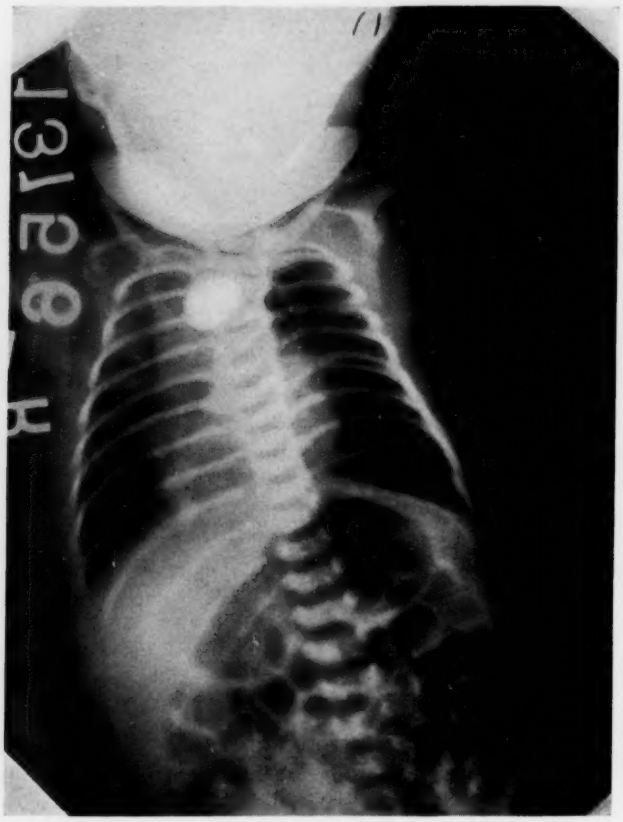


FIG. 3.—Roentgenogram of thorax and abdomen after the esophagus had been injected with lipiodol. The blind pouch of the esophagus is well defined, and the typical gaseous distention of the stomach and small intestine is well depicted (Case 1).

Operation.—Through a high paramedian left rectus incision the cardiac end of the esophagus was ligated with umbilical tape. A simple gastrostomy was performed by the technic of Kaader (Fig. 5). The infant was returned to the Drinker respirator where it remained until October 25 when it died from an aspiration pneumonia.

Autopsy Findings.—Esophageal atresia with agenesia and tracheo-esophageal fistula; bilateral aspiration pneumonia. The following associated congenital anomalies were present: Right kidney replaced by a large cyst; right ureter had a stricture at its lower end (Figs. 4, 5 and 6).

Case 2.—White female infant, born July 6, 1935, full term, weight 6.5 lbs. Physical examination revealed a normal female infant without any external evidence of congenital malformations. The infant continued to react normally until the first feeding, at which

Volume 103
Number 5

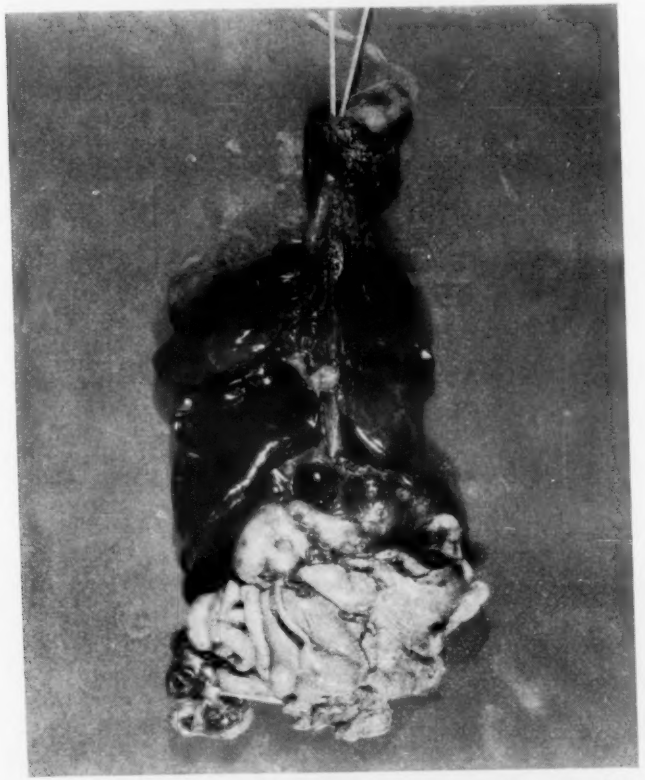


FIG. 4.—Photograph showing the entire viscera (Case 1). The blind pouch of the upper segment of the esophagus is well shown. The lower segment of the esophagus with ligature attached is seen beneath the liver. The gastrostomy tube is shown protruding from the anterior stomach wall.

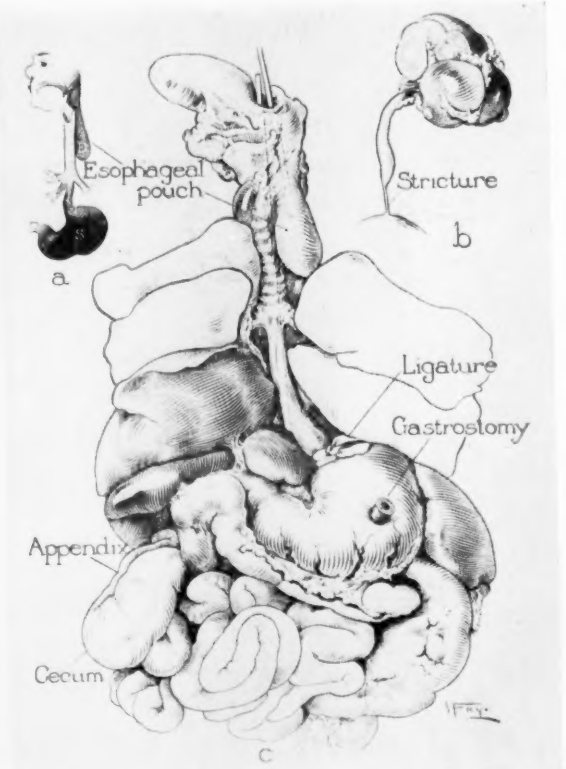


FIG. 5.—Drawing of the gross specimen shown in Fig. 4. The drawing shows: (a) the blind pouch of the esophagus and the tracheo-esophageal fistula; (b) the cyst of the right kidney and the stricture of the lower part of the right ureter; and (c) the anatomic relationships of the entire congenital anomaly with the ligature of umbilical tape around the cardiac end of the lower segment of the esophagus. The gastrostomy tube is seen projecting from the anterior wall of the stomach.

time she took fluid for a few seconds, which was soon regurgitated through both the mouth and the nose. Cyanosis occurred, the infant gasped for breath, the fluid ceased flowing from the nose and the mouth, the cyanosis disappeared, and the infant again appeared normal. This train of clinical signs and symptoms occurred at each feeding.

The infant was admitted to the Touro Infirmary on July 31, 1935, at 12:35 A.M. Examination at this time revealed an infant four days old who was apparently normal with the exception of a slight dehydration and elevation of the rectal temperature to 102° F.

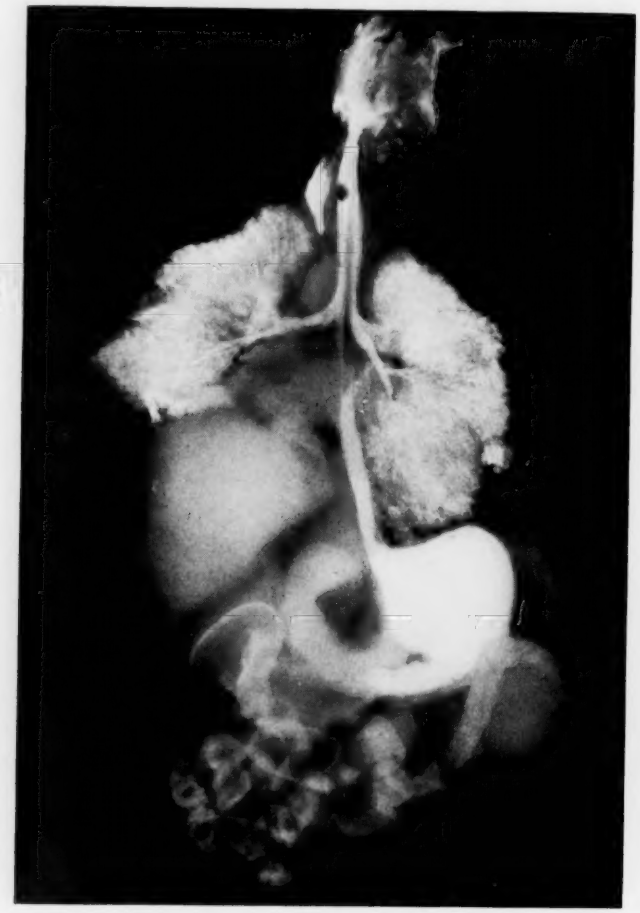


FIG. 6.—Roentgenogram of thoracic and abdominal viscera removed from Case 1, after the injection of barium into the trachea. The lungs, tracheo-esophageal fistula, trachea, esophagus, upper and lower segments, and stomach are visualized.

From the history a tentative diagnosis of esophageal atresia and tracheo-esophageal fistula was made. A small catheter was introduced into the esophagus where it encountered an obstruction about 12 cm. from the alveolar margin. Lipiodol was instilled through the catheter into the esophagus and a roentgenogram was made. This revealed a typical complete esophageal atresia and agenesia of the upper segment. Tracheo-esophageal fistula was also inferred because the stomach and small intestine were filled with gas (Fig. 7).

Operation.—July 31, 1935, under ether anesthesia the cardiac end of the esophagus

was ligated through a high left rectus paramedian incision. A gastrostomy was also performed by the technic of Ssabanejew-Frank.

The infant reacted well from the operation and was given fluids at regular intervals through the gastrostomy tube. On August 1, the infant suddenly became cyanotic and had great difficulty in breathing. The temperature was elevated to 102.8° F. Cyanosis and difficult respirations continued and the infant succumbed at 3:00 A.M. August 2, 1935.

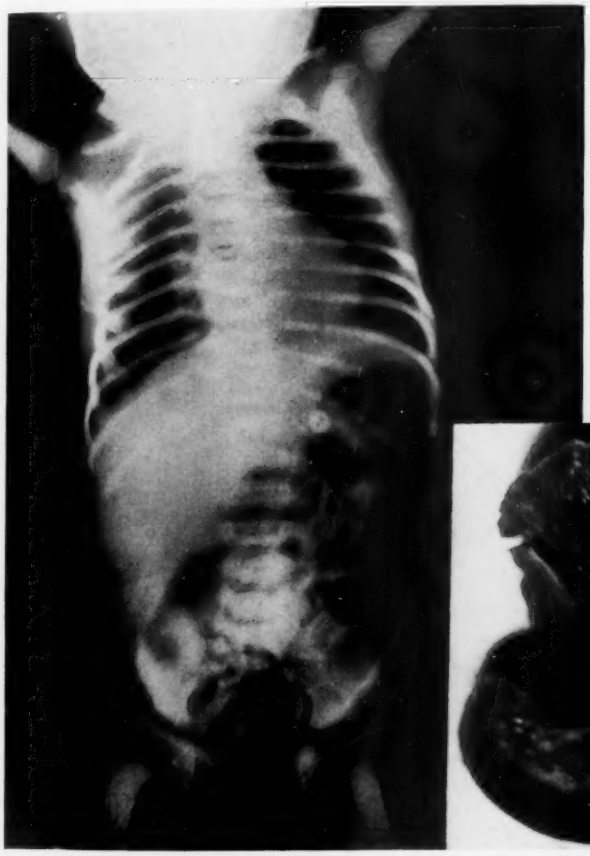


FIG. 7.—Roentgenogram of the thorax and abdomen (Case 2), showing the typical blind pouch of the upper segment of the esophagus in the upper thorax. The typical gaseous distention of both the stomach and the small intestine, so characteristic of tracheo-esophageal fistula, is well outlined.

FIG. 8.—Photograph of viscera removed from Case 2, showing tracheo-esophageal fistula opening just above the bifurcation of the trachea. The opening is opposite the point of the white marker.

Autopsy Findings.—Esophageal atresia with agenesia and a tracheo-esophageal fistula with the opening just above the bifurcation of the trachea (Fig. 8). The lungs revealed a bilateral aspiration pneumonia of both upper lobes. A horse-shoe kidney was an associated congenital anomaly.

REFERENCES

[1] Brennemann, J.: Congenital Atresia of the Esophagus with Report of Three Cases. Am. Jour. Dis. Child., vol. 5, p. 143, 1913.

[2] Strong, R. A., and Cummings, Harold: Congenital Atresia of Esophagus with Tracheo-esophageal Fistula. Am. Jour. Dis. Child., vol. 47, p. 1299, 1934.

[3] Mackenzie, M.: Malformations of the Esophagus. Arch. Laryngol., vol. 1, p. 301, 1880.
[4] Plass, E. D.: Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula. Johns Hopkins Hosp. Rep., vol. 18, p. 259, 1919.
[5] Hacker, V., and Lotheissen, G.: Chirurgie der speiseröhre. Neue Deutsche Chir., vol. 34, p. 192, 1926.
[6] Rosenthal, A. H.: Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula. Arch. Path., vol. 12, p. 756, 1931.
Rosenthal, A. H., and Himmelstein, U.: Congenital Atresia of the Esophagus with Tracheo-esophageal Fistula. Arch. Pediat., vol. 49, p. 444, 1932.
[7] Luschka: Cited by Rosenthal, Ref. 6.
[8] Klebs: Cited by Rosenthal, Ref. 6.
[9] Kreuter: Cited by Schridde, Ref. 10.
[10] Schridde, H.: Ueber Epithelproliferationen in der menschichen Speiseröhre. Virchows Arch. f. Path. Anat., vol. 191, p. 178, 1908.
[11] Forssner: Cited by Rosenthal, Ref. 6.
[12] Streeter: Cited by Rosenthal, Ref. 6.
[13] Lateiner, M.: Ein Fall von angeborener oesophagus-atresie mit Nachealkommunikation. Wien. klin. Wchnschr., vol. 22, p. 53, 1909.
[14] Rienhoff, W. F.: Congenital Arteriovenous Fistula. Bull. Johns Hopkins Hosp., vol. 25, p. 271, 1924.
[15] Kelly, A. B.: Congenital Abnormalities At or Near the Upper End of the Esophagus. Jour. Laryngol. and Otol., vol. 46, p. 521, 1931.
[16] Richter, H. M.: Congenital Atresia of the Esophagus: An Operation Designed for Its Cure. Surg., Gynec., and Obst., vol. 17, p. 397, 1913.
[17] Ssabanejew-Frank: Bickham's Operative Surgery, vol. 4, Philadelphia, W. B. Saunders Co., 1924.

DISCUSSION.—DR. URBAN MAES (New Orleans, La.).—I think I can add a small bit of encouragement to the very depressing report Doctor Gage has made, and at least point out a mistake that should not be made. These children usually come to the surgeon only after they have been given barium for roentgenologic examination, which is a serious and usually fatal error. I was discussing the subject a few weeks ago with Doctor Scott of the University of Rochester, and his experience parallels my own in the three cases I have seen personally—that all the children have pneumonia, and that the lung picture always shows a tiny fleck of barium in the center of each patch of pneumonia.

The diagnosis ought to be made in some other way. Usually the clinical history and physical findings are clear, and if they are not, and some visualization is necessary, the use of lipodol is less harmful than the use of barium.

Doctor Scott informed me that one of his patients lived six weeks and one three months after operation, which shows that something can be accomplished.

DR. CHARLES G. MIXTER (Boston, Mass.).—Doctor Gage's paper has been of great interest to me as for many years much of my work was devoted to the surgery of infants and children. He is to be congratulated on developing a technic that bids fair to offer a chance of success in this disastrous anomaly that up to the present time has invariably proved fatal.

He has spoken of the frequency of its occurrence. Our experience at the Children's Hospital in Boston entirely corroborates his statement. In the past ten years 23 instances of tracheo-esophageal fistula have been observed. We have classified esophageal malformations into two main groups, *i.e.*, (1) those in which the esophagus ends blindly and, (2) those in which there is a tracheal fistula. The second group may be subdivided into three classes.

Sixty per cent belong to the class in which the distal segment has a fistulous opening into the trachea, the lower end of the proximal segment ending blindly.

Up to 1929, a palliative gastrostomy was done. This did not accomplish its purpose as all cases invariably died of pneumonia, caused directly by the fistula or indirectly by aspiration of the contents of the blind esophageal segment. The site of the fistula has always been found just above the bifurcation of the trachea. In 1929, I made my first operative attack on the tracheo-esophageal fistula by a posterior approach through the posterior mediastinum on the right side. A considerable extent of two ribs at the level of the bifurcation were excised; by pushing the pleura laterally the region of the fistula was readily exposed. The right azygos vein usually crosses over the fistulous communication and this is dislocated and the opening severed and sutured. The separated distal segment of the esophagus is brought out and implanted in the incision in the back or in a stab wound. A catheter is placed through the esophagus to the stomach for feeding purposes. The first infant lived 57 hours following operation before succumbing to the pneumonia which was present before operation. After several futile attempts, it was found that distal esophagostomy was insufficient, as overflow of secretion from the blind proximal end was followed by a fatal pneumonia. Therefore, proximal esophagostomy through the neck, either at the primary operation or within a few days, was added. Nine operative attempts have been made in the last six years, with and without proximal esophagostomy. All have ended fatally, but in two instances the infants have lived for 15 and 30 days, respectively. It is, of course, a palliative procedure, but it is hoped that, if the infant survives, an esophageal reconstruction can be done later.

Dr. Vilray P. Blair (St. Louis, Mo.).—I do not wish to encourage surgical exercises, but Doctor Mixter said something that I think has some value. If you meditate a radical operative attempt, do it at the earliest possible moment and you will have a better chance than on the older child, if you have any chance at all.

Dr. Mims Gage (closing).—The object of presenting the subject of congenital tracheo-esophageal fistula was to stimulate discussion which may result in a more direct surgical attack on the lesion and thus lower the mortality rate and secure better end results.

Doctor Mixter's experience with these cases is of interest from the standpoint of his operative "transmediastinal approach." However, I do not believe that these patients can withstand any extreme dissection or manipulation. The surgical attack of the fistulous opening at its source is the ideal procedure, as it does not leave behind an esophageal pouch that will become infected and from which regurgitation into the tracheal tree will cause an acute pneumonitis. I agree with Doctor Blair's admonition that an infant can withstand more surgical trauma immediately after birth than it can several days later.

I should like to stress again the importance of keeping the upper esophageal pouch clean, in order to prevent filling and overflow with subsequent aspiration pneumonia. The upper esophagus should be mobilized to the outside as soon as possible. The lower segment must be drained by postural treatment with the head down until intratracheal manipulation, aspiration, and obliteration of the pouch can be accomplished.

ULCER OF THE PYLORIC SPHINCTER

J. Shelton Horsley, M.D.
Richmond, Va.

Ulcers within the grasp of the pyloric sphincter are uncommon. Their symptoms are often accentuated probably on account of their location. While the analogy is not entirely accurate because of the difference in the type of muscle and of nerve supply, the condition calls to mind a fissure in ano in which the discomfort and pain are caused not so much by the nature of the lesion as by its situation within the grasp of a band of muscle whose chief function is to remain tonically contracted, opening and closing at intervals under physiologic stimuli.

It is difficult to make a definite diagnosis of ulcer of the pyloric sphincter, because accurate localization of the lesion by roentgenology is often impossible. The intense spasm frequently overshadows the ulcer, though by fluoroscopic study and with numerous plates a roentgenologist can usually predict that an ulcer will be found somewhere in the region of the pylorus. Ulcers that originate on either the gastric or the duodenal side of the sphincter may touch or involve the tissue lining the pyloric sphincter, but these are not truly of the pyloric ring. According to Eusterman, the incidence of ulcers confined to the pyloric sphincter is not more than three or four per cent of peptic ulcers of all kinds. True ulcers of the pyloric ring must of necessity be gastric ulcers, because the gastric mucosa normally prolapses through the pyloric sphincter, and if histologic section shows an ulcer at the pyloric sphincter of duodenal tissue it cannot be classed as an ulcer of the pyloric ring.

While there is no distinct clinical entity in this group of cases, there are certain features which may suggest the probability of this type of ulcer. Eusterman mentions them as follows: "The comparatively brief duration of symptoms at the time the patient sought relief; the stormy course in the majority of cases, attributed to painful spastic manifestations; the pyloric obstruction of variable degree, resulting from spasm, inflammatory reaction or cicatrization; the dilatation of the stomach, even in the absence of consistent pyloric obstruction; the high volume of gastric content; the preponderance of ulcers in men, and the difficulty of clinical and roentgenologic diagnosis so far as actual localization of the lesion to the ring is concerned."

In the Proceedings of the Staff Meetings of the Mayo Clinic (vol. 10, p. 435, July 10, 1935), W. L. Butsch states that this lesion is comparatively rare—that only 46 cases of ulcer of the pyloric ring could be found in the surgical records of the Mayo Clinic for the last 15 years. Ten of these ulcers were excised and were found to be benign, and the pathologic diagnosis of the remaining 36 cases was not known. Eleven per cent of the cases in this series reported by Butsch showed apparent achlorhydria; the average value

for free hydrochloric acid was 36.5, which is 6.7 units below the normal level of free hydrochloric acid. In typical gastric ulcer free hydrochloric acid is only 5.4 units below the normal level. Usually there is some dilatation of the stomach with obstruction. The average age of the patients was 47 years, which is about the age of patients with gastric ulcer. Of the 46 cases, 41 were males and five were females.

The symptoms averaged about 6.1 years, though in seven cases "dyspepsia" had been present for 15 years, and if these were eliminated the average would be greatly reduced. Butsch states that the average duration of symptoms before the patient applied to the Mayo Clinic for examination is much shorter

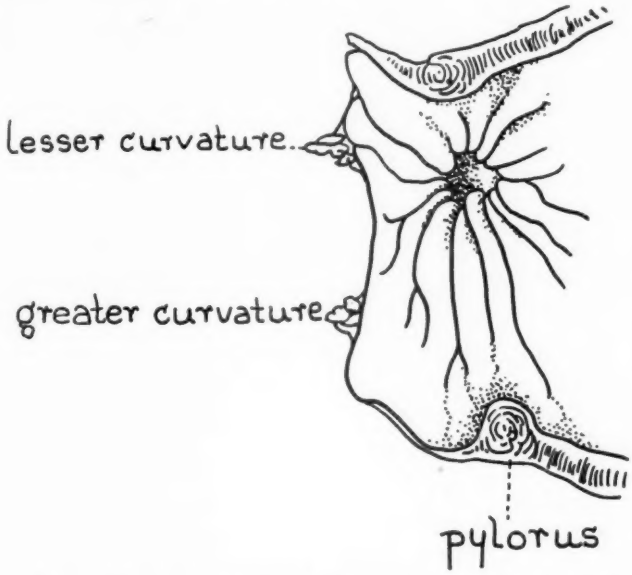


FIG. 1.—W. E. G., Surg. No. A-1565. White male, aged 36. First symptoms eight years ago, with violent attacks of pain in the stomach about 15 minutes after meals, followed by nausea and vomiting. He was relieved by diet in three weeks, and two months later had dyspepsia temporarily relieved by medication. Attacks were worse in the past three months. He lost 20 pounds in the past three years. The average free hydrochloric acid was 51.25. Operation, March 10, 1927. Appendectomy, cholecystectomy, and partial gastrectomy. The stomach specimen shows peptic ulcer in the pyloric sphincter, in the upper anterior wall.

in ulcer of the pyloric ring than in either duodenal or gastric ulcer, and he found that the significant symptomatology of this ulcer is that the duration of the symptoms is usually shorter and the course more stormy, with comparatively little relief by ordinary medical treatment.

Of the 46 cases reported from the Mayo Clinic for the last 15 years, resection of the pylorus and the adjacent portion of the stomach was done in only ten cases. In these there was a Billroth II type of partial gastrectomy. In the remaining 36 cases a posterior gastro-enterostomy was performed, and the diagnosis depended solely upon the observation of the surgeon. In many of these cases there were adhesions and perforation, which contra-indicated a partial gastrectomy. Consequently, it would seem impossible to establish

accurately the location of the ulcer in these 36 cases. Even when a partial gastrectomy has been done and the specimen opened and examined, the exact location of the lesion in its relation to the pyloric sphincter may sometimes be difficult.

During the past 11 years, from September 12, 1924, to October 23, 1935, I have operated upon 12 cases of peptic ulcer either solely or partly within the pyloric ring. Of those that were entirely within the pyloric ring there were nine cases. Of those that apparently originated elsewhere and extended to the pyloric ring there was one. Of cases within the pyloric ring and with ulcers elsewhere there were two. In all of these 12 cases a partial gastrectomy, a modification of Billroth I method, which has been described elsewhere, was done.

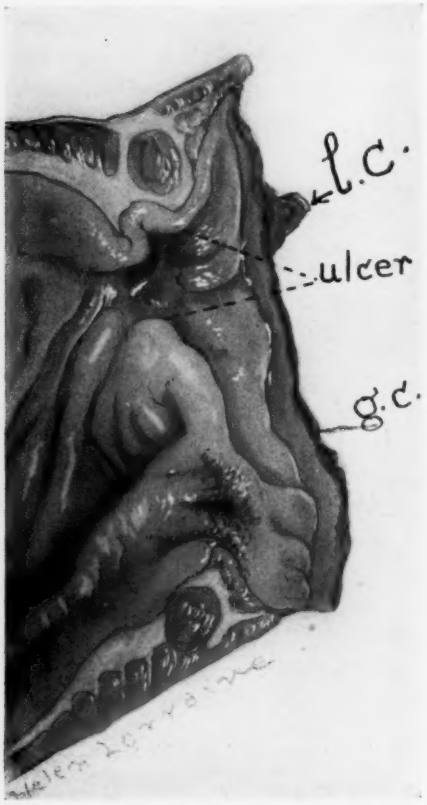


FIG. 2.—W. W. F., Surg. No. B-1455. White male, aged 46. Three months before admission the patient had severe pains in the epigastrium, relieved by food and alkalies. There were no previous symptoms, no nausea, vomiting, nor tarry stools. Average free hydrochloric acid was 75. Roentgenogram showed a filling defect on the gastric side of the pylorus. Operation, August 8, 1935. Partial gastrectomy and appendectomy. Specimen, opened behind, shows an ulcer within the grasp of the pyloric sphincter, and adjoining it is a superficial erosion, on the posterior side of the lesser curvature.

During this time I have done 173 operations for peptic ulcer of all kinds, so that the percentage of operations for ulcer in the pyloric ring to total operations for peptic ulcer is 6.94 per cent. This rather high percentage of ulcers in the pyloric ring in comparison with operations for peptic ulcers elsewhere should not be assumed to be the proper ratio of the actual incidence of this ulcer, because peptic ulcers in other locations respond more readily to medical treatment and often do not need operation.

It may be that during this period of 11 years there have been in my practice more ulcers within the pyloric sphincter than I am reporting in this paper. In operations in which, for instance, gastro-enterostomy was done, particularly if there was much inflammation about the region of the first part of the duodenum and the prepyloric portion of the stomach, there may have been a lesion within the grasp of the sphincter. However, it seems impossible to make an accurate diagnosis of ulcer in the pyloric sphincter, unless the pyloric end of the stomach has been excised and the position of the ulcer carefully noted in the specimen removed. Consequently, all the cases of ulcer within the pyloric

ring that are here reported are based upon the observation of the position of the ulcer in the specimen removed by a partial gastrectomy.

In two cases in which a description of the operation and the observation

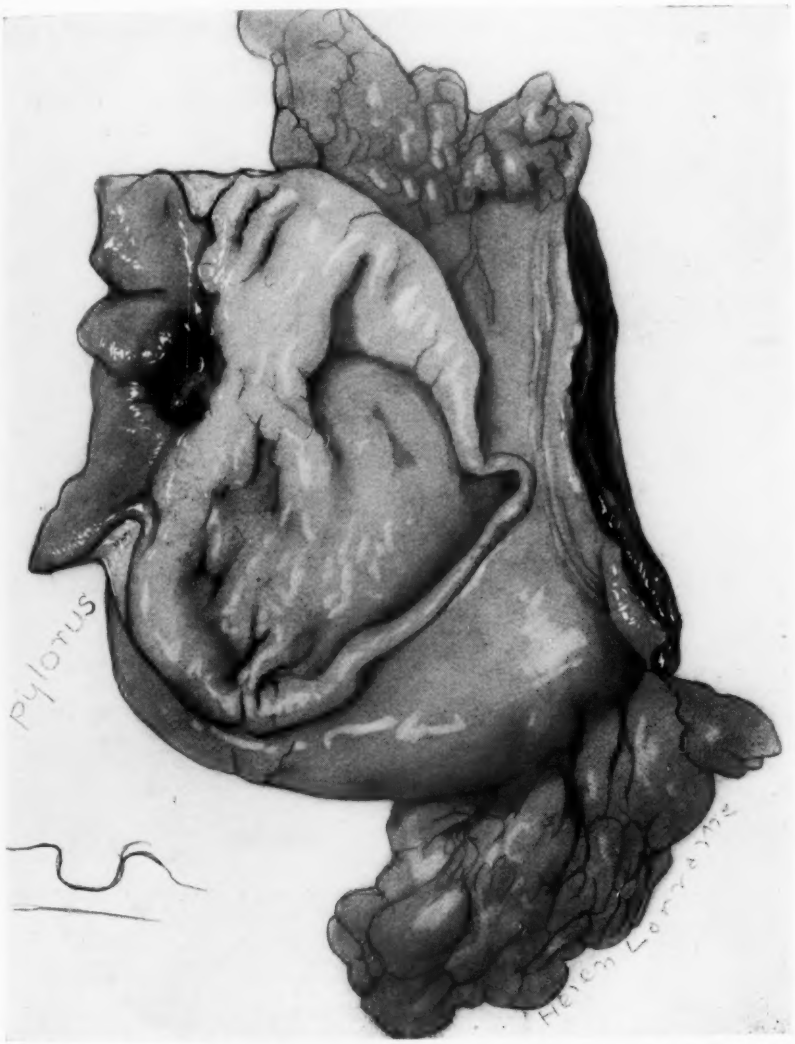


FIG. 3.—C. R. M., Surg. No. A-7311. White male, aged 42. A year before admission the patient had occasional cramps in the epigastrium after eating. This was accompanied by nausea and relieved by vomiting. The pain increased in frequency and severity until on admission the cramps and nausea followed everything ingested, but there was immediate relief by vomiting. He lost 21 pounds in the past three weeks. The average free hydrochloric acid was 51.25. Roentgenogram showed a penetrating ulcer of the pylorus. Operation, August 11, 1934. Appendectomy, resection of anterior gastric vagus nerves, and partial gastrectomy. Specimen shows a single rather deep ulcer in the grasp of the pyloric sphincter. Inset shows the depth of the ulcer. It was in the upper posterior portion, and the base of the ulcer involved pancreatic tissue.

of the lesion were dictated immediately after the partial gastrectomy, it was recorded that the ulcer was in the pyloric ring, and the specimen was preserved in formalin. Many months afterwards, when the specimen was again

examined, the ulcer appeared to be distinctly on the duodenal side. However, microscopic examination showed the surrounding tissue to be gastric mucosa (Broders). The ulcer which was not deep had been shoved through the sphincter by the hardening process of the formalin.

The symptoms are by no means similar in each case, and yet on the whole they seem to bear out the observations of Eusterman and Butsch, in the severity of the symptoms, and in the fact that relief by medical means is apparently more difficult than in ulcer either of the stomach higher up or of

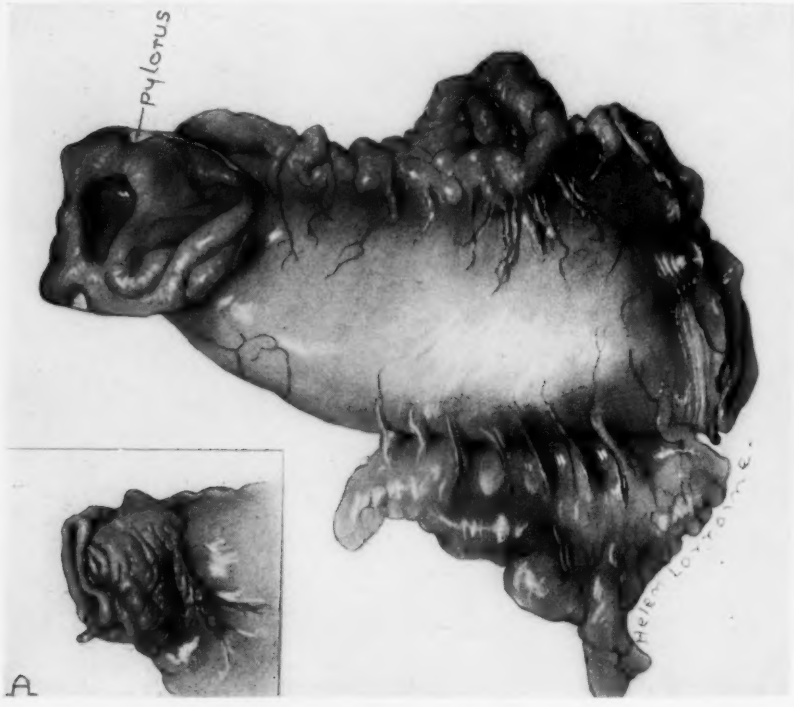


FIG. 4.—Mrs. M. B. H., Surg. No. A-7000. White female, aged 58. Had suffered from indigestion for the past 18 years. Seven years ago had an attack of pain in the gallbladder region, with nausea and vomiting. Two years ago an attack of weakness with nausea and vomiting. There was flatulence after meals. Twelve days before admission there was pain in the epigastrium, with fulness in the abdomen and nausea and vomiting. These symptoms continued at intervals up to admission. The abdomen was markedly tender in the upper right quadrant. It appeared to be an acute condition, possibly a slow penetration of an ulcer. The patient was of the ulcer type. There was no gastric analysis or roentgenologic examination. Operation, October 21, 1931. Partial gastrectomy. The specimen shows an ulcer within the grasp of the pyloric sphincter in the posterior portion which had penetrated into the pancreas. Inset shows a portion of the pancreas removed, which formed the base of the ulcer.

the duodenum. The symptoms may be periodic discomfort or hunger pain often only incompletely relieved by food, soda, or vomiting. Severe bleeding occurred in two of the cases. On account of the spasm, symptoms and sign of obstruction with late emptying of the stomach are common. The roentgenologic diagnosis, though not always positive, is helpful in that it shows unusual spasm, but without the filling defect or local deformity that is supposed to be characteristic of ulcer in the stomach or in the duodenum.

The ulcers may be penetrating, but even if they are not the amount of spasm and obstruction that occurs is very marked and often out of proportion to the small lesion. This is doubtless caused by the location of the ulcer.

The treatment for this condition is partial gastrectomy, using preferably a modification of the Billroth I type of operation that we have adopted during the past 12 years. The advantages of this procedure for this lesion are rather obvious. Even though the gastric acidity is not high as a rule in these cases,

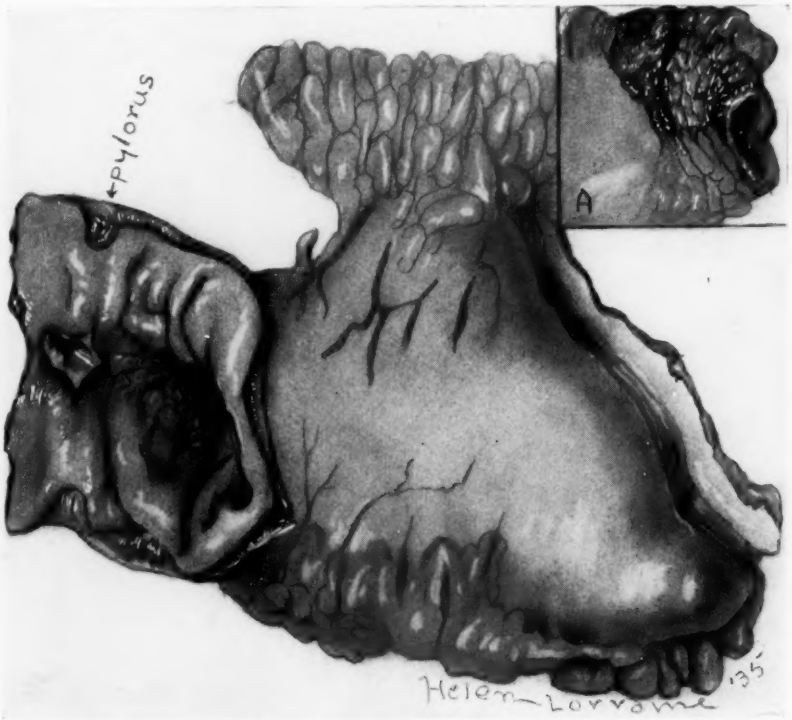


FIG. 5.—R. E. C., Surg. No. B-750. White male, aged 47. Twenty years ago the patient was jaundiced. Each year, usually in the fall, for the past 15 years, he had abdominal pain relieved by food and medication. These attacks increased in severity and in frequency, and in the past two years they had been accompanied by vomiting that was almost projectile. Twice he had formal medical treatment extending over some weeks. The last attack began six days before admission to the hospital, and was almost continuous to the time of admission. There were nausea and vomiting, and cramps so severe as to require hypodermics of morphine. He occasionally vomited blood, and lost some weight. The average free hydrochloric acid was 16.5. Roentgenologic examination showed a duodenal defect. Operation, January 10, 1935. Appendectomy, resection of the anterior gastric vagus nerves, and partial gastrectomy. Specimen shows a deep penetrating ulcer within the posterior portion of the grasp of the pyloric sphincter. Inset shows the portion of the pancreas which was removed, which formed the base of the ulcer.

which frequently suggests the probability of cancer, there is a tendency toward ulcer formation. In one of our cases there were ulcers elsewhere. It seems quite possible that some of these apparently solitary ulcers within the pyloric sphincter, are surviving lesions after ulcers in other portions of the stomach or duodenum have healed. They, too, doubtless would have healed but for the influence of the pyloric sphincter. At any rate, it would seem to be the part of wisdom to provide, as far as can be, against a recurrent ulcer, by having

the stomach discharge its contents into the duodenum instead of into the jejunum. As is well known, the sensitivity of the mucosa of the gastrointestinal tract to irritation from the gastric juice increases from the duodenum down to and including the large bowel. If, after the resection, the stomach is united to the duodenum and at the same time the normal relationship of the physiologically important lesser curvature with the upper border of the duodenum is maintained, not only will a cure probably be effected but a

FIG. 6.—C. C. B., Surg. No. A-771. White male, aged 47. The patient had bilious spells at rare intervals, and anorexia and indigestion for 10 or 12 years, but the attacks were never very severe. The day before admission, after a heavy meal, he was nauseated and vomited dark material which was probably blood. The following morning after breakfast he vomited large quantities of bright blood. He was admitted to the hospital later in the day. On account of the bleeding there was no gastric analysis nor roentgenologic study. May 17, 1926, five days after admission to the hospital, a partial gastrectomy and appendectomy were performed. Specimen shows two ulcers within the grasp of the pyloric sphincter, one on the posterior wall at the lesser curvature, and the other on the lower border.

restoration of the physiologic function of the stomach and duodenum will be more nearly accomplished than by other methods.

That partial gastrectomy is the proper treatment for this type of ulcer seems to be supported by a consideration of the clinical and pathologic features of this lesion. As has been mentioned, a true ulcer of the pyloric sphincter always has a background of pyloric (gastric) mucosa, because this mucosa normally lines the pyloric sphincter. An ulcer of the pyloric ring, then, is a gastric ulcer whose symptoms are usually accentuated.

Most of the ulcers in this series of cases have been on the posterior wall

of the sphincter, either in the center penetrating to the pancreas, or near the upper or lower borders of the stomach. In such cases though a pyloroplasty will expose the lesion which may, of course, be excised and the mucosa sutured, local excision and suturing of a gastric ulcer elsewhere in the stomach is often unsatisfactory. Dragstedt and Vaughn (Gastric Ulcer Studies, Arch. Surg., vol. 8, p. 791, 1924) have shown that one of the best methods of creating an experimental gastric ulcer in a dog is to place sutures in the pyloric portion of the gastric mucosa. A good treatment for an ulcer in the right half of the stomach is partial gastrectomy. This pyloric ring type of ulcer not only has the vices of a gastric ulcer elsewhere, but is peculiarly liable to hemorrhage. We had two cases of very severe hemorrhage in this series of 12 cases. It also often perforates and, like other gastric ulcers, may become malignant. Pyloroplasty is not by any means free from the occurrence of a late stricture. If a gastro-enterostomy is established, a jejunal ulcer may result, and this is worse than the original ulcer. If a pyloroplasty is performed, and there is a recurrent ulcer, a partial gastrectomy will be much more difficult than it would have been if a partial gastrectomy had been done in the first instance. As to the danger, while this series of cases is small (12) there were no fatalities.

In the 12 cases, which are being reported, a partial gastrectomy by a modification of the Billroth I type was done. In nine of the cases the lesion was solely within the pyloric sphincter; in one the ulcer invaded the sphincter from the duodenal side; in two cases there was also an ulcer elsewhere.

The average age of these 12 patients was 46.6 years. Two of them were women, and ten were men. The average duration of symptoms was 10¾ years, the duration of the symptoms varying from three months to 20 years. Several of the patients gave a long standing history of indigestion, but the severe symptoms were of only recent date. There was severe pain in nine cases, mild pain in one case, and two patients complained of no actual pain. In the latter two cases there was vomiting of much blood, and there was some bleeding in one other case. In nine cases there was a history of vomiting. A gastric analysis was done in ten of these cases. The average value for free hydrochloric acid was 32.9 units. The highest average value of free hydrochloric acid in any one case was 75. In two cases there was no free hydrochloric acid.

In ten cases a roentgenologic examination was made, and in three obstruction was present after 24 hours. One patient was operated upon immediately as an emergency on account of severe symptoms, and in one other patient, operated upon a few days after admission, there had been a severe hemorrhage from the stomach and no gastric analysis or roentgenologic examination was made. Six of these patients were obviously of the so called ulcer type; in four the type was unknown; in two unrecorded.

In one case the ulcer was partly within the pyloric sphincter, but chiefly on the duodenal side. The patient was a man, 47 years of age. The symptoms had extended over ten years, but were severe only recently. There had been no vomiting. Gastric analysis showed an achlorhydria. Roentgenologic ex-

amination showed retention after 24 hours. The patient was of the ulcer type. Two cases had multiple ulcers. The average age of these patients was 51.5 years. One was male, and the other female. The average duration of symptoms was 14.5 years. One had severe pain; the other had no pain, but acute hemorrhage. There was vomiting in one case. The average value of free hydrochloric acid was 27.1 units.

In the nine patients with ulcer entirely within the pyloric sphincter, there were eight men and one woman. The average age was 45.5 years. The average duration of symptoms was 9.9 years. There was severe pain in seven cases, mild pain in one, and no pain in one. There was vomiting in eight cases. Two patients had a history of bleeding, severe in one and only occasional slight bleeding in the other. Five of the patients were of the so called ulcer type. The average value of free hydrochloric acid was 39.25 units. There was no free hydrochloric acid in one case; the highest was in one patient in whom the value was 75 units. In one case the roentgenologic examination showed obstruction after 24 hours.

Dr. Douglas G. Chapman rendered valuable aid in the analysis of the symptoms of these cases.

MECKEL'S DIVERTICULUM WITH PEPTIC ULCER

DONNELL B. COBB, M.D., M.S.
GOLDSBORO, N. C.

MECKEL'S diverticulum is due to the persistence of a portion of the vitelline duct or yolk stalk. Being a rudimentary blind pouch on the intestinal tract, it is subject to the same disease processes which have brought fame and dishonor to the appendix—inflammation, gangrene and perforation.

Johann Meckel[46] (1781–1833) discovered this diverticulum and described its anatomic origin. In his description of the diverticulum (1812) Meckel states that "The structure of the inner layer is exactly the same as in the portion of the intestine from which the diverticulum springs." He evidently did not recognize that, at times, the diverticulum is lined entirely or in part by gastric mucosa and that it may be the seat of peptic ulcer formation with subsequent hemorrhage and perforation. Denecke,[16] in 1902, reported the first ulcer in Meckel's diverticulum but it remained for Deetz,[14] in 1907, to point out the occurrence of gastric mucosa in these cases and to stress the peptogenic character of these ulcerations.

Incidence.—The incidence of Meckel's diverticulum is difficult to determine. Because few surgeons make a routine search for its presence, operating room statistics are not accurate. At the Johns Hopkins Hospital, 15 cases were found in 2,600 necropsies, or .57 per cent, while Mitchell, of Chicago, in 1,635 autopsies reported 39 cases, or 2.38 per cent. From these combined figures the incidences can be placed at 1.2 per cent. It occurs twice as often in males as in females.

Peptic ulcer seems to be the most common complication of Meckel's diverticulum. Schaetz[63] in a careful study of 30 diverticula found that 50 per cent contained abnormal elements. Pancreatic tissue or mucosa resembling that of the duodenum or jejunum may be found within its lumen. He estimates the incidence of gastric mucosa at 16 per cent. Certainly all cases containing gastric mucosa are cases of potential ulcer with hemorrhage and perforation.

Huebschmann[34] compares the ulcers in Meckel's diverticulum to the gastrojejunal ulcers following gastro-enterostomy. In both instances the ulcerations occur at the junction of the gastric and intestinal mucosa. Schaaff[62] analyzed the fluid content of the diverticulum removed by him and found it to contain 40 free hydrochloric acid and 70 total acidity.

Lindau and Wulff[44] call attention to the fact that the secretion of hydrochloric acid from gastric mucosa contained in a Meckel's diverticulum "begins or increases synchronously with the activity of the stomach—this implies that the production of gastric juice in Meckel's diverticulum occurs at a time during which the small bowel is empty and when no neutralization takes place by food and intestinal juice. In such manner especially favorable

conditions are created for the generation of peptic lesions, a circumstance that we find borne out by the occurrence of these ulcers in Meckel's diverticulum even at an early age."

Besides this article by Lindau and Wulff other excellent articles dealing with peptic ulcer in Meckel's diverticulum have recently appeared by Mason and Graham,[45] and Johnston and Renner.[40] A review of the literature reveals 98 cases, including one previously published by the author. To this number I would like to add two more, making a total of 100.

CASE REPORTS

Case 1.—R. K., a white boy, aged 9, was first seen in 1931. At that time he had several severe exsanguinating intestinal hemorrhages, passing large amounts of light and dark blood. There had been no pain associated with this condition and his past history was negative for any abdominal discomfort or digestive disturbance. This boy was seen a short time after another patient had been operated upon for a perforated peptic ulcer of Meckel's diverticulum and with this case freshly in mind, a diagnosis was made of bleeding ulcer of Meckel's diverticulum and operation was advised. The parents refused operation, however, and took the child home.

He was seen again, one year later (1932). In the interim he had had no further intestinal hemorrhage and had had no abdominal discomfort. On this second visit he did complain of diffuse abdominal discomfort of 48 hours' duration. This discomfort had become localized in the right lower quadrant where he complained of much tenderness. There had been no nausea and vomiting. Physical examination revealed a well nourished and developed boy of 10 years. All findings were negative except marked tenderness and rigidity in the right lower quadrant. His temperature was 98.6° F.; pulse, 88; white blood count, 13,000 with 91 per cent neutrophils; the urinalysis was negative. From these findings a diagnosis was made of acute appendicitis associated with a peptic ulcer in a Meckel's diverticulum.

The patient was operated upon the same day, using a right midrectus incision. The appendix was found situated retrocecally and was very acutely inflamed. After the appendix was removed, exploration of the lower ileum showed, about 18 inches from the ileocecal valve, a Meckel's diverticulum situated on the antimesenteric border. The diverticulum was 6 cm. long and 2 cm. in diameter at its base, tapering to the tip, and was conical in shape. There was a fibrous cord, the size of a match, running from the tip of the diverticulum to the umbilicus (Fig. 1). The diverticulum was resected, removing a cuff of ileum. The adjacent mucous membrane of the ileum appeared normal. The opening in the ileum was closed transversally with two rows of Lembert sutures, and the abdominal incision was closed without drainage. The patient made an uneventful convalescence and was discharged from the hospital 12 days later.

Microscopic examination by Dr. J. B. Bullitt.—"The mucosa is thick and is thrown into a number of rugae. The surface epithelium is tall columnar, with clear cytoplasm and basal nuclei. Beneath this, there are fairly long tubular glands, lined with columnar epithelium which takes a fairly deep stain; between these columnar cells are a moderate number of globular cells with eosinophilic granules. This picture resembles the mucosa of the stomach. At one end of the specimen there is a small piece of the gut; and at the juncture of the two types of mucosa, there is a shallow ulcer, whose base is covered with a thin layer of necrotic débris (Fig. 1A). A very moderate amount of leukocytic exudate is about the ulcerated region, and a scanty leukocytic infiltration is seen everywhere throughout the specimen. The muscularis is scantily developed, consisting of merely occasional bundles of fibers imbedded in a thick layer of connective tissue."

Case 2.—J. P., a white male infant 1½ years old. This patient was brought in with the complaint of excruciating abdominal pain, and the passage of bloody stools. The

parents stated that the child had always seemed healthy. Four days before admission they noticed the passage of a large, dark, offensive stool, resembling old blood. The child had four such stools the same day. The following morning, or three days before admission, while eating breakfast, the child complained of intense pain in the abdomen, and that same morning there was another dark, offensive, bloody stool. The following

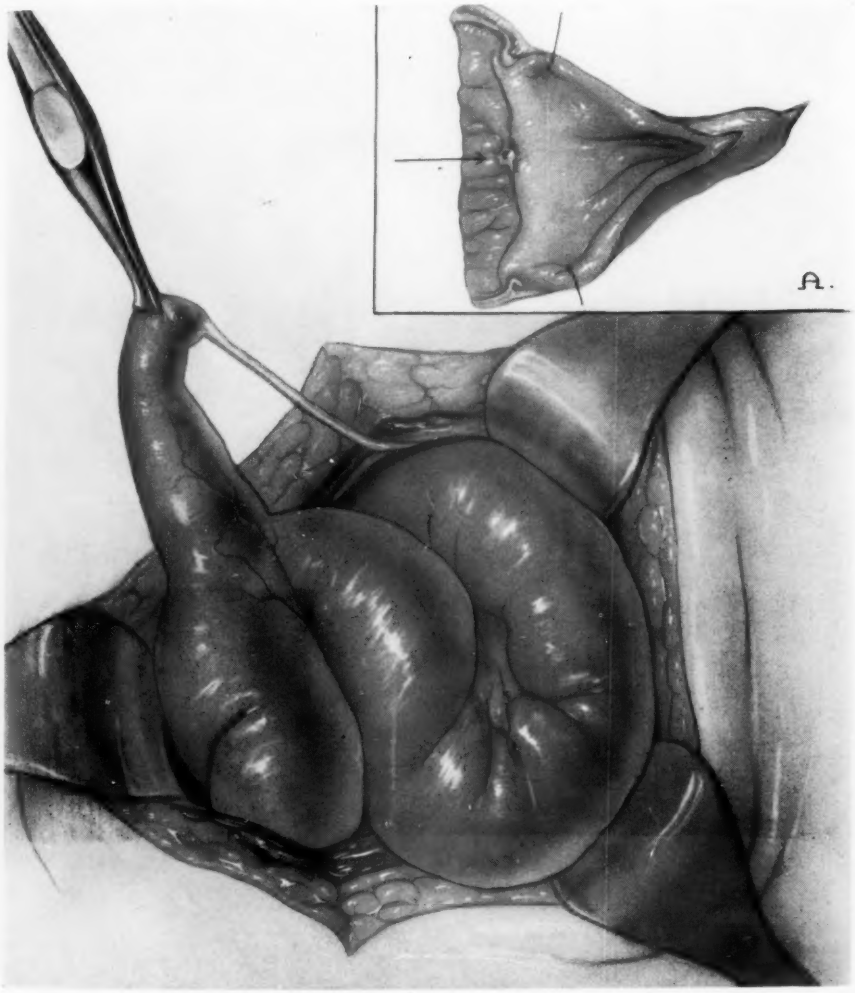


FIG. 1.—(Case 1.)—The Meckel's diverticulum as seen at operation presented no evidence of inflammation. Inset shows peptic ulcer at junction of gastric and intestinal mucosa. Gastric mucosa verified microscopically.

day the abdomen was somewhat swollen and the child began to vomit. The vomiting continued and the abdominal swelling gradually became more pronounced. A dose of castor oil was given the day the first bloody stool was noted, and again following the excruciating abdominal pain while eating breakfast on the next morning.

On examination the child was a well developed infant of 1½ years. He was in definite shock. The rectal temperature was 101.6° F., and the pulse was so thready and weak that it could not be counted. The abdomen was markedly and diffusely distended; it was tympanitic throughout. There were no palpable masses; there were no definite

TABLE I

ULCER CASES WITH MICROSCOPICALLY VERIFIED GASTRIC MUCOSA

Author	Year	Age	Sex	Symptoms: Pain	Symptoms: Intestinal Hemorrhage	Preoperative Diagnosis	Ulcer Perforated	Recovered	Died	Remarks
1. Hilgenreiner[33]	1903	18	M.	+	+	Appendicitis	−	+		
2. Deetz[14]	1907	9	M.	+	−	Appendicitis	+	+		
3. Huebschmann[34]	1913	4½	M.	+	+	Peritonitis	+		+	Diverticulum found at autopsy
4. Gramen[27]	1915	10	M.	+		Appendicitis	+	+		
5. Callender[7]	1915	19 mos.			+		−		+	Found at autopsy. Death from hemorrhage
6. Meulengrecht[19]	1918	12	M.	+	+		−		+	Death from other cause. Found at death
7. Mueller[54]	1919	11	M.	+	+	Peritonitis	+	+		
8. Megevaud and Dunant[47]	1922	28	M.	+	+	Duodenal or intestinal ulcer	−	+		
9. Brasser[3]	1924	15	M.	+	+	Tumor or polyp	+		+	Cause of bleeding not found at operation. Diverticulum at autopsy
10. Humbert[37]	1924	11 mos.	M.	+	+	Peritonitis	+		+	Diverticulum found at autopsy
11. Guibal[30]	1924	14	M.	+	+	Intestinal tuberculosis	−	+		
12. Pascale[57]	1925	41	F.	+	+	Intestinal ulcer	−	+		
13. Ulrich[75]	1925	8 mos.	M.	+	+	Peritonitis	+		+	
14. Abt and Strauss (Case 1)[1]	1926	2	F.	+	+		−	+		
15. Etchegorry[20]	1926	16	M.	+		Appendicitis	+	+		

15. Etchegorry[20]	1926	16	M.	+		Appendicitis	+		
16. Kleinschmidt[41]	1927	15	M.	+	+	Appendicitis	+	+	
17. Buechner[5]	1927	24	M.			Peritonitis	−	+	
18. McCalia[55]	1927	4	M.	+	+		+		+ Not operated. Found at autopsy
19. Jackson, A. S.[38]	1927	14	M.	+	+	Ulcer of Meckel's diverticulum	−	+	
20. Taylor[73]	1927	17 mos.	M.		+		−		+ Died of hemorrhage. Diverticulum found at autopsy
21. Gramen[27]	1927	21	M.	+		Peritonitis	Not stated	+	
22. Gramen[27]	1927	11	F.	+		Peritonitis	Not stated	+	
23. Meiss[18]	1928	2	M.		+	Intussusception or polyp	+	+	Diverticulum adherent to ileum into which ulcer had perforated
24. Hartglass[32]	1928	4	F.	+		Appendicitis	+	+	
25. Peterman and Seeger[59]	1928	6	M.	+	+	Ulcer of Meckel's	+	+	Operated first for appendicitis. Intestinal bleeding continued. Diverticulum found at 2nd operation
26. Franke[24]	1929					Peritonitis	+		+
27. Treplin[74]	1929					Appendicitis	−	+	
28. Winkelbauer[80]	1929	2	M.	+	+	Intussusception	−	+	
29. V. Haberer[31]	1930	21	M.		+	Bleeding duodenal ulcer	−	+	Two duodenal ulcers found at operation; also bleeding ulcer in diverticulum
30. V. Haberer[31]	1930	13	M.	+	+	Postappendectomy abscess	+	+	
31. Schmidt[64]	1930	18	M.			Peritonitis	+	+	Two ulcers found; one had perforated
32. Edwards[19]	1930	16	M.	+	+	Peritonitis	+	+	
33. Achsner and Karelitz[2]	1930	15 mos.	F.		+	Tumor	−	+	

TABLE I (*Continued*)

Author	Year	Age	Sex	Symptoms: Pain	Symptoms: Intestinal Hemorrhage	Preoperative Diagnosis	Ulcer Perforated	Recovered	Died	Remarks
34. Achsner and Karelitz[2]	1930	2	M.	+	+	Intussusception	−	+		
35. Fevre, Patel and Lepart[22]	1930	7	M.	+		Appendicitis	+	+		
36. Fevre, Patel and Lepart[22]	1930	5 mos.	M.	+	+	Intussusception	+	+		
37. Cobb[10]	1931	18	M.	+	+	Appendicitis	+	+		
38. Debre[13]	1931	17 mos.	M.	+	+		+		+	
39. Buechner[5]	1931	8	F.	+			−	+		
40. Tavernier and Guilleminet[72]	1931	10	F.		+	Possible ulcer of Meckel's diverticulum	−	+		
41. Lindau and Wulff[41]	1931	15	M.			Appendicitis				Operated upon for appendicitis. Diverticulum found at autopsy
42. Greenwald and Steiner[29]	1931	15 wks.	M.	+		Perforated ulcer of Meckel's diverticulum				
43. Greenwald and Steiner[29]	1931	10	M.	+	+		+	+		
44. Hudson and Koplik (Case 12)[35]	1932	5½	M.	+	+	Peritonitis	+		+	
45. Hudson and Koplik (Case 31)[35]	1932	5½	M.	+	+		−	+		
46. Vaughn and Singer[77]	1932	7	M.	+		Appendicitis	+	+		
47. Mason and Graham[45]	1932	9 mos.	M.	−	+		−	+		

47. Mason and Graham[45]	1932	9 mos.	M.	−	+		−	+	
48. Goetsch[26]	1932	19	M.			Intestinal obstruction	Healed	+	Ulcer had perforated and had drained 6 yrs. previously
49. Burger[6]	1932	7	M.	+	−	Ileus	+	+	
50. Royster[61]	1932	7	M.	+	+	Exploratory	+	+	
51. Marty and Roudil[60]	1932	7	M.	+		Appendicitis	+		+
52. Wulff[81]	1932	16	F.				−	+	
53. Schaaff[62]	1932	7	M.	+	+	Perforated ulcer of Meckel's diverticulum	+	+	Diverticulum 60 cm. long, contained a fluid with 40 free HCl and 70 total acidity
54. Hudson[36]	1933	11	M.	+	+		−		+
55. Hudson[36]	1933	6 mos.	M.	+	+	Peritonitis	+		+
56. Dragstedt[18]	1933	17 mos.	M.	+	+	Intestinal ulcer	−	+	
57. Johnston and Renner[10]	1933	18 mos.	M.	+		Appendicitis	+	+	
58. Johnston and Renner[10]	1933	15 mos.	F.		+	Ulcer of Meckel's diverticulum	−	+	
59. Clark[9]	1933	8	M.	+	+		−	+	Multiple erosions
60. Lamy and Mondor[52]	1933	4½	M.	+	−	Appendicitis	+	Not mentioned	
61. Del Carril[15]	1934	2	M.	+	+		−	+	
62. Weeder[79]	1934	9 mos.	F.	+	+	Perforated ulcer of Meckel's diverticulum	+	+	
63. Deton and Huard (Case 1)[17]	1935	18	M.	+	+	Ulcer of Meckel's diverticulum	−	+	
64. Deton and Huard (Case 2)[17]	1935	11	M.	+	+	Ulcer of Meckel's diverticulum	−	+	
65. Croes and Van Hasselt[11]	1935	3½	M.	+	+		−	+	
66. Cobb (See text)	1935	9	M.	+	+	Ulcer of Meckel's diverticulum	−	+	

TABLE II

ULCER CASES. MUCOSA NOT INVESTIGATED

Author	Year	Age	Sex	Symptoms: Pain	Symptoms: Intestinal Hemorrhage	Preoperative Diagnosis	Ulcer Perforated	Recovered	Died	Remarks
1. Denecke[16]	1902	7	M.	+		Appendicitis	+		+	
2. Brentano[4]	1904	24	M.	+		Appendicitis	+	+		
3. Lawen[43]	1909	23	M.	+		Peritonitis	+	+		
4. Stern[69]	1917	5½	F.	+		Peritonitis	+	+		Large bowel mucosa
5. Stern[69]	1917	3	M.	+	+	Peritonitis	+	+		
6. Griffith[28]	1914	19 mos.	M.	+	+		+		+	Found at autopsy
7. Schreuder[65]	1923	8	M.	+	+	Duodenal ulcer (roentgen ray)	−	+		
8. Jackson, R. H.[39]	1924	10	M.	+	+	Surgical abdomen	−	+		
9. Stulz and Woringer (Case 1)[71]	1925	4 mos.	M.	+	+	Ileus, peritonitis	+		+	
10. Stulz and Woringer (Case 2)[71]	1925	11 mos.	M.	+	+		+		+	Found at autopsy
11. Abt and Strauss (Case 2)[1]	1926	1 mo.	M.	+	+		−	+		
12. Fuss[25]	1926	37	F.	+	+	Bleeding gastric ulcer	−	+		
13. Moll[51]	1926	5 mos.			+	Intestinal polyp	−		+	Found at autopsy
14. Kleinschmidt[41]	1927	45	M.	+	+	Tumor	+	+		
15. Shannon[67]	1928	18 mos.	F.		+	Ulcer of Meckel's diverticulum	+	+		
16. Winkelbauer[80]	1929	19 mos.	M.	+	+	Intussusception	−	+		No gastric mucosa on microscopic examination

17. Schwarz and Daly[66]	1929	8	M.	+	+	Bleeding ulcer of Meckel's diverticulum	−	+		
18. Smith and Hill[68]	1929	14 mos.	M.	+	+	Intestinal obstruction	+	+		
19. Christie (Case 3)[8]	1931	3 wks.	M.				—			Found at autopsy
20. Christie (Case 10)[8]	1931	2 wks.	M.				—			Found at autopsy
21. David[12]	1932	30	M.	+	+		+	+		Ulceration may have been carcinomatous
22. Finney[23]	1932	29	M.	−	+	Ulcer of Meckel's diverticulum	−	+		No gastric mucosa found
23. McKeen[56]	1932	53	M.	+	+	Duodenal ulcer (roentgen ray)	−	+		No gastric mucosa on microscopic examination
24. Walters and Wilkins[78]	1932	20	M.	+	+	Intussusception	+		+	
25. Pouget and Tavernier[72]	1933	5	F.	+	+	Ulcer of Meckel's diverticulum	+	+		
26. Fellows[21]	1933	19	M.	+	−	Appendicitis	+	Not stated		
27. Miller and Wallace[50]	1933	17	M.	+	+	Acute obstruction	+	+		
28. Valer, F.[76]	1934	8	M.	+	−	Peritonitis	+		+	No gastric mucosa on microscopic examination
29. Montgomery (Case 1)[53]	1935	13 mos.	M.	+	+	Ulcer of Meckel's diverticulum	−	+		
30. Montgomery (Case 2)[53]	1935	10 mos.	M.	−	+	Intussusception	−	+		
31. Montgomery (Case 3)[53]	1935	16 mos.	M.	+	+	Ulcer of Meckel's diverticulum	+	+		
32. Montgomery (Case 4)[53]	1935	11 mos.	M.	+	+	Ulcer of Meckel's diverticulum	+	+		
33. Pasman[58]	1935			+	+	Intussusception	+	Not stated		
34. Cobb (See text)	1935	18 mos.	M.	+	+	Peritonitis	+		+	No gastric mucosa on microscopic examination

areas of rigidity, but there was a sense of increased resistance in the right lower quadrant. The child was put to bed, treated for shock, and dextrose in Ringer's solution was administered. Eight hours later, the child's condition being slightly improved, an exploratory operation was decided upon. A positive preoperative diagnosis had not been made, but it was felt that the child had had some rupture of a hollow viscus. The operation was performed through a right rectus incision. On entering the peritoneal cavity there was much free fluid and some intestinal contents. Examination of the appendix showed it to be mildly inflamed at its tip; it was not ruptured and did not appear responsible for the symptoms. Further exploration showed numerous adhesions of the ileum to the anterior abdominal wall just below the umbilicus. On separating these adhesions a

FIG. 2.—(Case 2.)—Punched-out perforation at margin of diverticulum. Gastric mucosa not found microscopically.

Meckel's diverticulum was disclosed. It could not be determined just how far from the ileocecal valve the diverticulum was situated. It arose from the antimesenteric border of the ileum, it was conical in shape, being 2.5 cm. in diameter at its base and 1.5 cm. at its tip, and about 4 cm. in length. At the junction of the diverticulum and ileum there was a punched-out perforated ulcer (Fig. 2). From this perforation was exuding a large amount of intestinal contents. The diverticulum was resected, removing a portion of the ileum. The opening in the ileum was closed transversely with two rows of Lembert sutures. No further exploration was done. The peritoneal cavity was washed out with ether, and two Penrose drains were placed in the pelvis. During the operation the patient received continuous intravenous dextrose in Ringer's solution, 500 cc. being given. Following the operation the child failed to react and died three hours later.

Pathologic examination by Dr. C. C. Carpenter.—"Gross description.—Specimen consists of a flattened piece of tissue that is 3.2 by 2.5 by .6 cm. To one side of the center is an oval perforation 0.3 cm. in diameter. The perforation is surrounded by a fibrinopurulent exudate. The lining consists of unbroken mucosa except for the point of perforation. The walls of the perforation are smooth and clean cut. Microscopic description.—Section shows the wall of the intestine, near the center of which there is a rounded perforation. The wall of the perforated area shows considerable necrosis and many neutrophils and lymphocytes. The mucosa for a considerable distance outside of the necrosed area shows necrosis with the submucosa and muscular layers preserved. An acute inflammatory reaction is seen throughout the section."

One hundred cases have been reviewed and are tabulated in two groups. Table I contains 66 cases of ulcer in which gastric mucosa was demonstrated microscopically. Table II contains 34 cases of ulcer in which no microscopic examination was made, or in which no gastric mucosa was found. In the few cases in which no gastric mucosa was found, it is entirely possible that the microscopic search was not sufficiently thorough or the island of gastric mucosa that had been present was destroyed by the process of ulceration and perforation. In any event the symptomatology and the gross findings clearly belong to the peptic ulcer group. The two groups, therefore are considered together. It is upon a study of these 100 cases of peptic ulcer in Meckel's diverticulum that this paper is based.

Age.—This disease is essentially one of infancy and childhood. Although the ages in this series of cases ranged from two weeks to 53 years, the great majority occurred in early childhood. Seventy-four per cent were found in the first 15 years of life (Table III).

TABLE III

AGE DISTRIBUTION

From birth to fifth year	40	74%
Five to ten years	23	
Ten to fifteen years	11	
Over fifteen years	23	
Age not stated	3	
Total	100	

Youngest, 2 wks.
Oldest, 53 yrs.

Sex.—It has been noted that Meckel's diverticulum occurs twice as frequently in males. There is, however, a far greater preponderance of ulcer formation in males. Eighty-one of these ulcer cases occurred in males and 14 cases in females. Thus the sex distribution approximates that of duodenal ulcer and congenital hypertrophic pyloric stenosis. In five cases the sex was not stated (Table IV).

Pain.—There is usually some associated abdominal pain. This symptom was complained of in 80 per cent of these cases. The pain does not always have the typical text-book location in the right lower quadrant with radiation

TABLE IV

SEX DISTRIBUTION

Males	81
Females	14
Sex not stated	5
Total	100

to the umbilicus. It is not unusual for the discomfort to closely simulate that of duodenal ulcer, being situated in the epigastrium, and may be periodic or postprandial in nature, as in the cases of Megevaud,[47] Huebschmann[34] and the one previously reported by the author.[10] At times, the pains are colicky in nature. These periodic, postprandial or colicky pains usually occur in the adult cases. In many instances, however, and especially is this true in children, there is no history of pain until the occurrence of perforation. Pain, of course, is much more frequent in the perforated cases, having occurred in 51 of the 55 cases, but it was also complained of in 29 of the 43 non-perforated cases.

Hemorrhage.—Intestinal hemorrhage is the most frequent complication. The bleeding may be profuse and in the non-perforated cases reported by Callender[7] and Taylor,[73] it was the direct cause of death. The blood may be bright red but usually it will have undergone partial decomposition within the intestine and appear as a black, tarry stool, as though the hemorrhage had taken place high up in the intestinal tract. Mason and Graham[45] have previously emphasized the importance of intestinal hemorrhage as a diagnostic sign and have called attention to the fact that the bleeding may extend, intermittently, over a period of months or years, often being traceable to infancy or very early childhood. Intestinal bleeding was present in 72 of these 100 cases. It occurred with equal frequency in the perforated and non-perforated groups (Table V).

TABLE V

INCIDENCE OF PAIN AND INTESTINAL HEMORRHAGE AND THE MORTALITY IN PERFORATED AND NON-PERFORATED ULCERS IN MECKEL'S DIVERTICULA

	No.	Pain	Intestinal Hemorrhage	Died	Mortality Percentage
Perforated ulcers	55	51	36	20	36
Non-perforated ulcers	43	29	36	5	12
Perforation not stated	2				
Total	100	80	72	25	27

Perforation.—Next to hemorrhage, the most frequent, and the most serious complication in peptic ulcers of Meckel's diverticulum is perforation. This occurred in 55 of the 100 collected cases.

It is not definitely known why ulcers of Meckel's diverticulum have such

a strong tendency to perforate. It is true, however, that the walls of these diverticula are often poorly formed, containing, at times, but little musculature, and thus form a much thinner base for the ulcer than is the case in gastric or duodenal ulcer.

That perforation is an extremely dangerous complication is shown by the fact that 20 of these 55 cases died, a mortality of 36 per cent. No stronger argument could exist for an earnest attempt at early diagnosis and early operation, before the perforation has occurred. The contents of the lower portion of the ileum being more infective than that of the stomach and duodenum, it is natural to suppose that a perforation in this location would be very dangerous. In the 43 non-perforated cases there were five deaths, a mortality of 12 per cent. Four of these cases, however, were not recognized during life but were found at autopsy. About the fifth case no particulars are given. In the cases that were operated upon before perforation had taken place there were no deaths.

Diagnosis.—The correct preoperative diagnosis of peptic ulcer of Meckel's diverticulum must necessarily be made by the process of exclusion. It is essential, of course, that the possibility be kept in mind. During recent years the literature has contained an increasing number of articles dealing with this condition and as a result of this a correct diagnosis has been made with increasing frequency. The more common causes of abdominal pain and melena, such as gastric and duodenal ulcer, polyps, malignancy, intussusception, a low grade ulcerative colitis and hemorrhoids must first be excluded.

TABLE VI

PREOPERATIVE DIAGNOSES

Ulcer of Meckel's diverticulum	17
Appendicitis	18
Peritonitis	18
Intussusception or obstruction	10
Duodenal or gastric ulcer	5
Intestinal tumor or polyp	4
Acute abdomen	3
Intestinal ulcer or tuberculosis	3
Ileus	1
Not stated	21
Total	100

The 17 cases correctly diagnosed were reported since 1927, six of them during 1935.

The diagnosis was first correctly made by Dr. E. A. Ketterer, of Montport, Wis., in 1927. He had seen a similar case previously. Since then, a total of 17 correct preoperative diagnoses have been made, six of which were during 1935.

In the non-perforated cases there were nine correct diagnoses. Their ages ranged from 13 months to 29 years. All of these cases passed large amounts

of blood, many of them intermittently over a period of months or years. Most of them complained of some abdominal pain. This was either colicky in nature and closely related to the time of bleeding or it was periodic and postprandial in character, resembling duodenal or gastric ulcer. Such a diagnosis is justifiable only after excluding the more common causes of melena and abdominal pain. When, however, no apparent cause can be found for such symptoms, an exploration is justified in order that an ulcerated diverticulum might be found and removed before perforation has taken place.

In the perforated group there were seven correct diagnoses. Their ages ranged from seven months to seven years. All of these cases, too, had passed large amounts of blood by rectum. Six of them had much abdominal pain. (In one case this was not stated.) It is true that the great majority of the

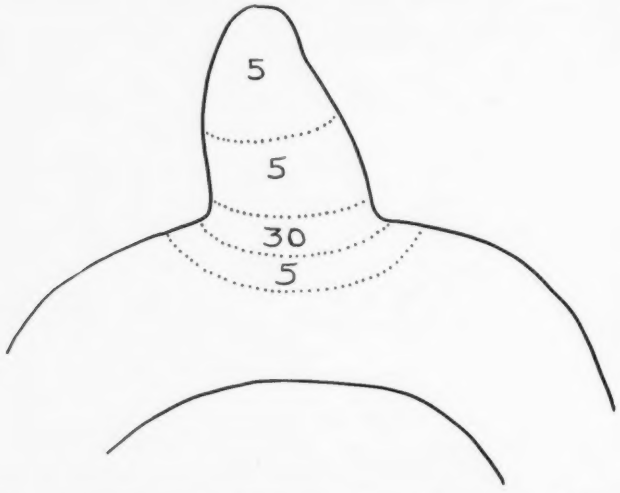


FIG. 3.—Diagram showing distribution of ulcers in 45 cases in which this was specified.

perforated cases were operated upon for appendicitis, peritonitis, intussusception or obstruction. However, whenever there is a so called "acute abdomen" associated with or preceded by the passage of a large amount of blood by bowel, exploration is demanded and a careful search for a Meckel's diverticulum should be made. (In one case in which a correct diagnosis was made it was not stated whether perforation had taken place.)

And so, in final analysis, the diagnosis of peptic ulcer in a Meckel's diverticulum, whether perforated or not, must depend upon the passage of large bloody stools and abdominal pain of variable degree. It should be remembered, however, that pain may be entirely lacking. When none of the more common causes which produce such symptoms can be found, an ulcerated diverticulum must be considered and operation is indicated. Roentgenologic examination has proven to be of practically no diagnostic assistance.

Treatment.—The procedure of choice in these cases is resection of the diverticulum with a portion of the adjacent ileum. Because the gastric mucosa

may occupy any portion of the diverticulum and because the ulcerations occur at the junction of the gastric and intestinal mucosa, these ulcers are situated in various parts of the diverticulum, *i.e.*, in the tip, neck or base of it, or outside of the diverticulum and in the ileum proper. Figure 3 shows the various locations of the ulcer in the 45 cases in which this was stated. Because so many of them occur at the base of the diverticulum and a few out in the ileum, it is always advisable to remove a cuff of ileum about the diverticulum when doing a resection.

Comment.—Unquestionably numerous cases of Meckel's diverticulum with ulceration have been encountered and operated upon that have not appeared in the literature. Meckel's diverticulum is no longer considered a surgical curiosity and if the whole truth were known a peptic ulcer in this location would probably not be a great rarity.

In all operations upon the gastro-intestinal tract, when the symptoms are not readily accounted for by the findings, a search should be made for a Meckel's diverticulum, particularly in children. Especially is this so when operating for appendicitis and when that organ is not found to be sufficiently inflamed to account for the symptoms. If this is done, the cause of many cases of so called idiopathic hemorrhage may be discovered, instances of unexplained peritonitis will be fewer, and many perforations of the diverticulum will be avoided.

Because any Meckel's diverticulum possesses numerous possibilities for serious trouble, and because a peptic ulcer in the location has such a strong tendency to perforate, resulting in a high mortality, any Meckel's diverticulum found at operation should be removed whenever possible.

BIBLIOGRAPHY

[1] Abt, I. A., and Strauss, A. A.: Meckel's Diverticulum as a Cause of Intestinal Hemorrhage. J.A.M.A., vol. 87, p. 991, 1926.

[2] Achsner, P. W., and Karelitz, S.: Peptic Ulcer of Meckel's Diverticulum and Ileum. ANNALS OF SURGERY, vol. 91, p. 573, 1930.

[3] Brasser, A.: Ulcus Pepticum Perforans des Meckel's-chen Divertikels. Zentralbl. f. Chir., vol. 51, p. 2423, 1924.

[4] Brentano: Entzuendung and Perforation eines Meckelschen Divertikels mit nachfolgender Peritonitis. Deutsche med. Wchnschr., vol. 30, p. 1301, 1904.

[5] Buechner: Die Histologie der pept. Veraenderungen und ihrer Beziehungen zum Magencarcinom. Jena: Gustav Fischer, 1927. (Cited by Lindau and Wulff.)

[6] Burger, W.: Ueber das Meckelsche Divertikel mit Magenschleimhaut. (Thesis) 18, p. 80, Heidelberg, 1932.

[7] Callender, G. R.: Gastric Glands in Meckel's Diverticulum. Am. Jour. Med. Sci., vol. 150, p. 69, 1915.

[8] Christie, A.: Meckel's Diverticulum; pathologic Study of 63 Cases. Am. Jour. Dis. Child., vol. 42, pp. 544–553, September, 1931.

[9] Clark, D. F.: Hemorrhagic Meckel's Diverticulum. Am. Jour. Surg., vol. 22, pp. 308–311, 1933.

[10] Cobb, D. B.: Perforated Ulcer of Meckel's Diverticulum. ANNALS OF SURGERY, vol. 94, p. 256, 1931.

[11] Croes, F., and van Hasselt, J. A.: Hemorrhage in Peptic Ulcer of Meckel's Diverticulum. Ned. tdschr. geneesk., vol. 79, pp. 3637–3639, 1935.

[12] David, V. C.: Discussion. Annals of Surgery, vol. 96, No. 5, pp. 893–910, November, 1932.

[13] Debre, R., Boppe, H., and Semelaigne, G.: Ulcere peptique du diverticule de Meckel. Perforation et fistule diverticulo-colique. Evolution subaigue des manifestations cliniques. Bull. Soc. de pediat. de Paris, vol. 29, pp. 402–412, July, 1931.

[14] Deetz, E.: Perforationsperitonitis von einem Darmdivertikel mit Magenschleimhautbau ausgehend. Deutsche Ztschr. f. Chir., vol. 88, p. 482, 1907.

[15] Del Carril, M. J., and Diaz Bobillo, I.: Sobre un caso de ulcera del diverticulo de Meckel. Prensa med. argent., vol. 46, pp. 3456–3459; also vol. 21, pp. 851–853, 1934–1935.

[16] Denecke: Ueber die Entzuendung des Meckelschen Divertikels und die Gangraen desselben. Deutsche Ztschr. f. Chir., vol. 62, p. 523, 1902.

[17] Deton: Ulcere peptique du diverticule de Meckel diagnostique avant la perforation. Huard, S.: Ulcere peptique du diverticule de Meckel diagnostique des la premiere hemorrhagie. Bull. Soc. nat. chir., Par., vol. 61, pp. 559–565, April 10, 1935.

[18] Dragstedt, L. R.: Ulcus Acidum of Meckel's Diverticulum. J.A.M.A., vol. 101, p. 20, 1933.

[19] Edwards, H.: Congenital Diverticula of the Intestine: with the Report of a Case Exhibiting Heterotopia. Brit. Jour. Surg., vol. 17, No. 65, pp. 7–21, 1930.

[20] Etchegorry, J.: Perforación de ulcera simple del diverticule de Meckel. Operación. Curación. Ann. de la Fac. de Med., vol. 11, p. 621, 1926.

[21] Fellows, F. M.: Inflamed Meckel's Diverticulum with Perforated Ulcer of Ileum. Brit. Med. Jour., vol. 2, p. 150, 1933.

[22] Fevre, Patel, and Lepart: Ulcus Perfores due diverticule de Meckel. Bull. et mem. Soc. Nat. de chir., vol. 56, p. 756, 1930.

[23] Finney, J. M. T.: Discussion. Annals of Surgery, vol. 96, No. 5, pp. 893–910, November, 1932.

[24] Franke, A.: Peptisches Ulcus in Meckelschen Divertikel. Arch. f. klin. Chir., vol. 157, p. 131, 1929.

[25] Fuss, H.: Ueber einen Fall von blutenden Ulcus in einem Meckelschen Divertikel. Deutsche Ztschr. f. Chir., vol. 199, p. 336, 1926.

[26] Goetsch, E.: Discussion. Annals of Surgery, vol. 96, No. 5, pp. 893–910, November, 1932.

[27] Gramen: Chronischen Ulcus in einem Meckelschen Divertikel mit Perforation und diffuser Peritonitis. Nord. Med. Ark. Kirurgi., vol. 48, pp. 1–7, 1915.

[28] Griffith, J. P. C.: Diseases Connected with Meckel's Diverticulum with Especial Reference to Diverticulitis. J.A.M.A., vol. 62, p. 1624, 1914.

[29] Greenwald, H. M., and Steiner, M.: Meckel's Diverticulum in Infancy and Childhood. Am. Jour. Dis. Child., vol. 42, p. 1176, 1931.

[30] Guibal, L.: Ulcere peptique d'un diverticule de Meckel provoquant des hemorragies intestinales profuses: operation: guerison. Bull. et mem. Soc. Nat. de chir., vol. 50, p. 349, 1924.

[31] Haberer, H. V.: Beobachtungen ueber Komplikationen von section der Meckelschen Divertikels. Deutsche Ztschr. f. Chir., vols. 225–226, p. 131, 1930.

[32] Hartglass, M.: Perforation d'un Ulcere Peptique siegeant sur un diverticule de Meckel. Operation. Guerison. Bull. et mem. Soc. nat. de chir., vol. 54, p. 1091, 1928.

[33] Hilgenreiner, H.: Entzuendung und Gangraen des Meckel'schen Divertikels. Beitr. z. klin. Chir., vol. 40, p. 99, 1903.

[34] Huebschmann: Spaetperforation eines Meckel-schen Divertikels nach Trauma. Muenchen. med. Wchnschr., vol. 60, p. 2051, 1913.

[35] Hudson, H. W., Jr., and Koplik, L. H.: Meckel's Diverticulum in Children. New England Jour. Med., vol. 206, p. 827, 1932.

[36] Hudson, H. W., Jr.: Meckel's Diverticulum in Children. New England Jour. Med., vol. 208, p. 525, 1933.
[37] Humbert, J.: L'Ulcere Peptique du Diverticule de Meckel. These. de doct., Paris, No. 395, 1924.
[38] Jackson, A. S.: Ulcer of Meckel's Diverticulum as a Cause of Intestinal Hemorrhage. ANNALS OF SURGERY, vol. 87, p. 252, 1927.
[39] Jackson, R. H.: Hemorrhagic Ulcer of Meckel's Diverticulum. ANNALS OF SURGERY, vol. 80, p. 222, 1924.
[40] Johnston, L. B., and Renner, G.: Peptic Ulcer of Meckel's Diverticulum. Surg., Gynec., and Obst., vol. 59, No. 2, pp. 198–209, August, 1934.
[41] Kleinschmidt, K.: Das Ulcus Pepticum des Meckel'schen Divertikels. Beitr. z. klin. Chir., vol. 138, p. 715, 1927.
[42] Koch, M.: Naturforscherversammlung in Munster, 1912. (Cited by Huebschmann.)
[43] Lawen: Perforiertes Meckelsches Divertikel. Munchen med. Wchnschr., vol. 56, pp. 478–479, 1909.
[44] Lindau, A., and Wulff, H.: The Peptic Genesis of Gastric and Duodenal Ulcer, Especially in the Light of Ulcers in Meckel's Diverticulum and the Postoperative Ulcers in the Jejunum. Surg., Gynec., and Obst., vol. 53, p. 621, 1931.
[45] Mason, J. M., and Graham, G. S.: Ulceration of Aberrant Gastric Mucosa in Meckel's Diverticulum, a Source of Intestinal Hemorrhage. ANNALS OF SURGERY, vol. 96, p. 893, 1932.
[46] Meckel, J. F.: Handbuch er pathologischen Anatomie (3 vols.), from vol. 1, pp. 555–597, Leipzig, 1812–1818.
[47] Megevaud, E., and Dunant, R.: Ulcere Peptique du diverticule de Meckel. Hemorragies intestinales. Rev. de chir., vol. 60, p. 536, 1932.
[48] Meiss, W. C.: Grave Hemorrhage from Ulcer in Meckel's Diverticulum. Nederl. Tijdschr. v. Geneesk., vol. 2, p. 4020, 1928.
[49] Meulengrecht, E.: Ein teilweise mit Magenschleimhaut bekleidetes und den Sitz eines Ulcus pepticum bildendes Meckelsches Divertikel. Arch. f. path. Anat., vol. 25, p. 125, 1918. (Cited by Stulz and Woringer.)
[50] Miller, R. H., and Wallace, R. H.: Meckel's Diverticulum in Acute Abdominal Emergencies. Tr. Am. Surg. Assn., vol. 51, pp. 234–243, 1933; also ANNALS OF SURGERY, vol. 98, pp. 713–721, 1933.
[51] Moll, H. H.: Giant Meckel's Diverticulum. Brit. Jour. Surg., vol. 14, p. 176, 1926–1927.
[52] Mondor, H. A., and Lamy, M.: Etude clinique des ulceres due diverticule de Meckel. J. de chir., vol. 41, pp. 553–584, April, 1933.
[53] Montgomery, A. H.: Tr. Western Surg. Assn., vol. 42, p. 295, 1932.
[54] Mueller, P.: Ueber des Ulcus pepticum (perforans) des persistierenden Dotterganges (Meckel'schen Divertikels) und seine Verwandtschaft mit den Ulcus Ventriculi. Beitr. z. klin. Chir., vol. 115, p. 560, 1919.
[55] McCalia, A. I.: A Case of Perforated Peptic Ulcer of Meckel's Diverticulum. Canadian Med. Assn. Jour., vol. 17, p. 79, 1927.
[56] McKeen, H. R.: Bleeding Ulcer of Meckel's Diverticulum. Colorado Med., vol. 29, p. 258, 1932.
[57] Pascale, G.: Peptic Ulcer in Meckel's Diverticulum. Riforma med., vol. 41, p. 721, 1925.
[58] Pasman: Oclusión intestinal por diverticulo de Meckel. Bol. Soc. cir. B. Aires, vol. 19, pp. 428–436, July, 1935.
[59] Peterman, M. G., and Seeger, S. J.: Meckel's Diverticulum with Hemorrhage. Am. Jour. Dis. Child., vol. 36, p. 515, 1928.
[60] Roudil, G., and Marty, J.: Sur un cas de perforation d'ulcere du diverticule de Meckel. Bordeaux Chir., No. 2, p. 179, 1932.

[61] Royster, H. A.: Discussion. ANNALS OF SURGERY, vol. 96, No. 5, pp. 893–910, November, 1932.
[62] Schaaf, G.: Beitrag zur Bildung des Ulcus pepticum im Meckelschen Divertikel. Deutsch. Ztschr. f. Chir., vol. 238, pp. 78–87, 1932.
[63] Schaetz, G.: Beitrage zur morphologie des Meckelschen Divertikels. Beitr. z. path. Anat., vol. 74, p. 115, 1925.
[64] Schmidt, L.: Perforation eines in einem Meckelschen Divertikel sitzenden peptischen Ulcus. Klin. Wchnschr., No. 32, p. 1523, 1930.
[65] Schreuder, O.: Ein seltener Fall Von Darmblutung. Zentral. f. d. ges. Chir., vol. 30, p. 490, 1925.
[66] Schwarz, E. G., and Daly, J.: Meckel's Diverticulum, Associated with Severe Hemorrhage. J.A.M.A., vol. 93, p. 1468, 1929.
[67] Shannon, W. R.: Meckel's Diverticulum in Infancy and Childhood. Arch. Pediat., vol. 45, p. 693, 1928.
[68] Smith, J. T., and Hill, O. W.: An Interesting Case of Meckel's Diverticulum, Associated with Intestinal Hemorrhage, Diverticulitis, Acute Intestinal Obstruction with Recovery. Arch. Pediat., vol. 46, p. 521, 1929.
[69] Stern, K.: Ueber Perforation des Meckelschen Divertikels. Deutsch. Ztschr. f. Chir., vol. 140, p. 343, 1917.
[70] Stone, J. B.: Discussion of paper of Abt and Strauss.[1]
[71] Stulz, E., and Woringer, P.: L'ulcere Peptique du Diverticule de Meckel chez L'enfant. Paris Med., vol. 2, p. 387, 1925.
[72] Tavernier, L., and Guilleminet, M.: Ulcere peptique du Diverticule de Meckel avec hemorragies graves. Lyon chir., vol. 29, p. 229, 1932.
[73] Taylor, A. L.: The Epithelial Heterotopias of the Alimentary Tract. J. Path. and Bacteriol., vol. 30, p. 415, 1927.
[74] Treplin, L.: Magenschleimhautinseln in Meckelschen Diverticel mit Ulcus Pepticum in Verbindungsstueck. Munchen. med. Wchnschr., vol. 76, p. 263, 1929.
[75] Ulrich, G. R.: Et Tilfaelde Af Perforeret Meckel's Diverticulum. Ugesk. f. Laeger., vol. 87, p. 664, 1925.
[76] Valer, F.: Perforation eines Meckelschen Divertikels. Arch. Kinderh., vol. 104, pp. 218–225, 1934–1935.
[77] Vaughn, R. T., and Singer, H. A.: Perforated Peptic Ulcer of Meckel's Diverticulum. ANNALS OF SURGERY, vol. 96, p. 230, 1932.
[78] Walters, G. A. B., and Wilkins, E. G.: Surgical Emergencies Due to Meckel's Diverticulum, illustrated by 6 cases. Lancet, vol. 2, pp. 1430–1432, December 31, 1932.
[79] Weeder, S. D.: ANNALS OF SURGERY, vol. 100, No. 3, pp. 550–553, 1934.
[80] Winkelbauer, A.: Ueber die chirurgischen Erkrankungen des Meckelschen Divertikels. Wien. klin. Wchnschr., vol. 42, p. 989, 1929.
[81] Wulff, H.: Zur Frage der peptischen Geschwuere in Meckelschen Divertikel. Chirurg., vol. 4, p. 926, 1932.

DISCUSSION OF THE PAPERS OF DOCTORS HORSLEY AND COBB

DR. JAMES M. MASON (Birmingham, Ala.).—As a Meckel's diverticulum is present only in 1.5 to 2 per cent of individuals, diseased condition of it will be only infrequently encountered. If it were present in 100 per cent of individuals, as is the appendix, it would probably have a position of greater importance in abdominal surgery than does that organ. Christie, from a study of 63 autopsies, classified three main types:

(1) The umbilical fistula, 6.3 per cent.

(2) Partial obliteration, with fibrous bands running from tip to umbilicus or some adjacent structure, 10 per cent.

(3) Typical diverticulum given off from the antimesenteric border of the

ileum, lying free in the peritoneal cavity and presenting a closed distal extremity, 82.5 per cent.

Two other rare types are noted:

(4) Giant diverticula of bizarre form or shape, sometimes coming off from the mesenteric border of the intestine and developing between the leaves of the mesentery.

(5) The umbilical polyp either attached to the remains of the omphalomesenteric duct or entirely cut off from internal connections.

In types 1 and 5 the diagnosis is made by inspection. Type 2 causes the greatest number of obstructions. Type 3 gives rise to intussusception, volvulus and diverticulitis and is the type in which peptic ulcer is most frequently observed.

Deetz, in 1907, seems to have been the first to suggest the peptic nature of an ulcer in Meckel's diverticulum, when he removed a perforated diverticulum and found that it contained gastric mucosa. Though Hilgenreiner, in 1903, reported a case of intestinal hemorrhage from an ulcer in Meckel's diverticulum, he did not recognize its peptic nature; and Callender, in 1911, made the first observation of a bleeding ulcer of Meckel's diverticulum in which the presence of gastric mucosa was definitely confirmed by histologic examination.

Up to 1924, some seven cases of intestinal hemorrhage from Meckel's diverticula had been reported at irregular intervals; and between 1924 and 1932, some 26 additional cases had been reported.

It is probable that interest was stimulated by the studies of Schaetz who made serial sections of 30 diverticula, and found gastric mucosa in 23.3 per cent.

Since no series of symptoms points definitely to the presence of a Meckel's diverticulum, the possible relation of intestinal hemorrhage to ulceration of Meckel's diverticulum was a real diagnostic discovery.

When analyses of reported cases show that, in almost every instance, hemorrhage preceded perforation by considerable intervals of time, it gives the thoughtful surgeon an opportunity to explore a case of obscure abdominal discomfort before perforation of an ulcer has taken place, and with a mortality of zero, whereas in those cases operated upon after perforation showed a mortality of 43 per cent. I was much impressed by a paper by Hudson and Koplik, in 1932, in which they reported 32 cases of Meckel's diverticulum in whom 63 per cent showed intestinal hemorrhage, and in whom hemorrhage was the chief symptom in seven instances.

DR. THOMAS S. CULLEN (Baltimore, Md.).—For many years I have been greatly interested in remnants of omphalomesenteric duct. Sometimes when the cord comes away, a little red raspberry-like body is seen in the umbilical depression. If this yields to astringents it is granulation tissue, if it is firm and little affected by astringents, it is usually the persisting outer end of the omphalomesenteric duct. When cut away, a small, spurting vessel has to be controlled. The outer surface of this raspberry-like mass is covered by mucosa, identical with that of the small bowel, while the central portion consists of non-striped muscle.

Occasionally the omphalomesenteric duct persists throughout its entire extent. In such cases, when the cord comes away, a fecal fistula exists at the umbilicus. The intra-abdominal portion of the omphalomesenteric duct may be represented as a Meckel's diverticulum which extends to the umbilicus; by a Meckel's diverticulum of variable length with a free end or with a small cord extending from the end of the diverticulum to the umbilicus. In some instances a thin solid cord extends from the mesentery, across the small bowel,

to the umbilicus. This is the remnant of the vessels of an atrophied Meckel's diverticulum.

Occasionally a piece of pancreas is embedded in the tip of a diverticulum. In one case I reported the presence of gastric mucosa in the umbilical depression outside of the abdomen and independent of the abdominal contents. When the patient ate, gastric juice was secreted at the umbilicus. I have never noted gastric mucosa at or near the base of a Meckel's diverticulum. Doctor Cobb's observations are most valuable as well as most instructive.

One point I should like to emphasize—where a raspberry-like mass exists in the umbilical depression, and where this is covered over by intestinal mucosa, then the parents should be told that if at any subsequent time the child has the slightest sign of intestinal obstruction that the abdomen should at once be opened and a Meckel's diverticulum looked for.

As a matter of fact, I have grown to have a very great respect for a Meckel's diverticulum. In all cases of appendicitis where the findings at operation do not tally with the severity of the symptoms, I not only carefully examine the contents of the pelvis, but also examine for a Meckel's diverticulum.

Dr. J. M. T. Finney (Baltimore, Md.).—Doctor Cobb's historic résumé is especially interesting. It is always well to know something of the medical and surgical worthies whose names are on our lips all the time, but about whom we know little or nothing. My interest in Meckel's diverticulum has been great since the first instance that I met with, which happened to be the first supposed appendicitis case upon whom I had ever operated. It had been diagnosed as an appendiceal abscess. The appendix, however, was found to be perfectly normal, but upon looking further a Meckel's diverticulum was discovered which presented a picture very similar to a traditional perforated appendix. Since that time, I have seen a number of similar cases. I can confirm the points that have been made, but will not take time to refer to all of them. I have seen such a relatively large number of Meckel's diverticula, that I now make it a routine practice to examine for them. Since we have been doing this routinely at the Union Memorial Hospital, we have been surprised at the number of cases we have found without symptoms.

Doctor Cobb called attention to the fact that this was usually a disease of early life, and was not a particularly fatal condition, hence a considerable number of these young people grow up, and it is found in a surprisingly high percentage of adults. I think we have reached the point where Meckel's diverticulum must be considered in every differential abdominal diagnosis, especially where hemorrhage and indefinite pain are present. I have noticed in one or two cases pain in the back, which has not been otherwise accounted for. We have not always made a positive diagnosis.

I was also interested in Doctor Horsley's presentation. I am not quite prepared, however, to be as radical as he is in performing partial gastric resections in these cases, and have always been partial to the pyloroplasty operation. Doctor Horsley has suggested an analogy to an anal fissure. In that case, if you thoroughly stretch the sphincter, you relieve the symptoms, which are due chiefly to muscular spasm, due to reflex irritation from the fissure. It seems to me that the same thing applies largely to ulcers of the pylorus. If you will put the pyloric muscle out of commission by excision of the ulcer and a segment of the pyloric muscle, the result will be satisfactory. I have just finished a case in whom a pyloroplasty was performed and a deep ulcer which was present excised, down to its floor. Sometimes one cannot excise the entire floor of the ulcer; we then treat it as we used to

treat old ulcers of the leg, by scarification, and then cover it over with mucosa. I have had the opportunity of observing some of these cases later pathologically. At any rate, you get a symptomatic cure of the symptoms, which is what interests the patient. I have found in the majority of instances that this has been all that is necessary. In a certain percentage of them, if after you have the pylorus open, you feel that you cannot reasonably expect a satisfactory result from the pyloroplasty alone, you can then proceed as Doctor Horsley has suggested.

Dr. George A. Hendon (Louisville, Ky.).—I think it would very well represent the highest peak of refinement of clinical diagnosis to be able to distinguish between a pylorus that reaches out and grasps an ulcer, and an ulcer that in its progress invades the pylorus. I have met with some experiences that have changed my ideas considerably in regard to the treatment of peptic ulcer. I have now a series of 60 cases that have been treated upon the principle of complete and perfect rest of the stomach, which is made possible by a method of intravenous nutrition which I have devised. The patients treated in this way have remained well for periods up to seven years. Doctor Mayo used to say that it was time to operate when a patient had had about 12 medical recoveries. We now cure our ulcer patients after they have had at least four surgical recoveries.

I wish to say a word or two about gastro-enterostomy. I have found that when gastro-enterostomy works well the ulcer is transferred to the jejunum. When it does not work well the ulcer in the pylorus or duodenum remains as it was at the start. It is much like the old country doctor said at our state meeting. Someone asked him how he was and he said, "I am getting along pretty well, but I have a bad case of piles and a pretty bad rupture, and if I push the piles in the rupture comes out and if I push the rupture in the piles come out."

In my group of 60 cases I can report 90 per cent recoveries. My experience in this field bears out the importance of rest which was emphasized in Doctor Horsley's opening remarks. In one case a man had his gallbladder removed and a few rearrangements of his interior decorations. I put him on the rest treatment for ten days and he got so well cured that the cicatricial tissue growing in the ulcer area produced a complete obstruction of the pylorus. I did a gastro-enterostomy and he gained 75 pounds and has remained well ever since.

Dr. Richard H. Miller (Boston, Mass.).—In looking up our cases with acute abdominal symptoms at the Massachusetts General Hospital, we found 15 adults who did not present symptoms of ulcer. They were all diagnosed as acute appendicitis or strangulated hernia. In two of them an acutely inflamed diverticulum was present in the hernial sac.

I agree with Doctors Cobb and Finney that in any case of obscure, vague abdominal pain we should search for these diverticula. In every case of melena in young people the presumptive diagnosis of Meckel's diverticulum is sound. I believe, thirdly, that in every instance of acute intestinal obstruction in a young person, without an abdominal scar, the diagnosis of Meckel's diverticulum is reasonable.

Dr. John C. A. Gerster (New York, N. Y.).—As to the statement about rest, at a meeting of the New York and Philadelphia Surgical Societies we reported two series of cases; one treated by bed rest in cases of ulcer of the lesser curvature, progress in which could be controlled by roentgenograms, and the other group placed on the Sippy diet. They ran about even. Some

have felt that putting a Levine tube in past the ulcer, and in that way putting the region at rest for a long time, was a good idea. Everyone admits that there are recurrences. The gastro-enterostomized patients may have relief for nine years and during the tenth develop a marginal ulcer. At the Harvey Society meeting last spring the director of the Cancer Research Section of the Middlesex Hospital in London stated that they had been able to obtain an extract from the middle lobe of the pituitary which if injected gave very good results.

Dr. J. Shelton Horsley (closing).—Doubtless my analogy of ulcer in the pyloric sphincter and fissure in ano is not entirely a happy one. It does tend to explain in a broad way the spasm, but a fissure in ano is open to inspection and treatment, whereas the other condition is not. Another thing is the marked tendency to recurrence in ulcer of the pyloric sphincter. There is also a tendency to bleed. In three of the cases referred to there were extensive hemorrhages. The ulcers on the posterior surface are very likely to erode the larger vessels. Pyloroplasty is a valuable operation but has a limited field. These are gastric ulcers, not duodenal. They are subject to cancerous change as well as recurrence. You may remember the work of Dragstedt and Vaughn in which they showed that the best way to produce a peptic ulcer in dogs was to put numerous sutures in the pyloric mucosa. In my early cases of pyloroplasty I sutured the pyloric mucosa and found quite a large percentage of recurrences at that point. The fact that these are gastric and not duodenal ulcers, that there is a marked tendency to bleed, to recur, and to undergo malignant change would seem to me to constitute definite indications for a type of gastrectomy that removes the tissues in this area, and preserves the function of the duodenum. There was no mortality in these cases.

OBSTRUCTION OF THE SMALL INTESTINE DUE TO FOOD PRODUCTS

F. Webb Griffith, A.M., M.D.
Asheville, N. C.

Elliot, in 1932, in reviewing the literature on this subject, stated that "Intestinal obstruction due to food is not a rare occurrence. A study of the available literature has revealed 39 cases reported since 1914, and all but ten in Germany or Austria. This is apparently traceable to the increased consumption of coarse foods in these countries as a result of the food shortage due to the World War. Only four instances have been reported in the United States during this time, and, as will be seen later, the diagnosis was not beyond question."

This paper is concerned with an analysis of these 39 instances in an attempt to elucidate the pathogenesis of this condition and to emphasize its importance. A case is reported of obstruction due to collards, proved by operation.

Of the 40 cases mentioned by Elliot, 13 were merely presumptive as they were not proven by operation or autopsy, and most of them recovered under conservative treatment. Of the remaining 27, five are eliminated because the obstruction was in the large bowel and therefore does not come under the scope of this paper, leaving 22 cases of obstruction of the small bowel due to food products.

In going through the literature I have found five others, which with my own makes six, or a total of 28 proven cases of impaction in the small intestine by food. Under the term food has been included bran, and also poppy seeds, which, as Elliot stated, were used in Germany to make soup and so called "ammunition" or "war" bread. The six cases not included in Elliot's list are, briefly, as follows:

(1) Roldan, of Montevideo, in 1929, reported a case of obstruction in a man aged 48, who had no upper teeth. A dried whole peach was removed from the ileum. Recovery.

(2) Herr, of Basel, in 1931, reported obstruction of the ileum by a single fig in a man aged 45. Ileostomy. Recovery.

(3) Davis, of Nashville, in 1931, removed a mass of bran the size of a hen's egg from the terminal ileum. Recovery.

(4) Zielke, of Leipzig, in 1933, operated for release of obstruction due to poppy seeds. Death.

(5) Block, of Philadelphia, in 1933, removed a mass of orange pulp from the lower ileum followed by enterostomy. Death from pneumonia ten days later.

(6) The author's case.

The list of foods which have been reported as causing obstruction comprises mushrooms, figs, beans, poppy seeds, grapes, bran, orange pulp, "ammunition bread," oats, corn, popcorn, gooseberries, cherries (with stones), raisins, sauerkraut, potatoes, a mixture of peas and potatoes, a mixture of peas and fruit, a whole dried peach, collards. Mushrooms headed the list as the cause in five instances.

In the 28 cases of proven impaction in the small bowel, pathology of the intestine, which might act as a predisposing cause, was found in only three cases. In one there was a tuberculous stricture in the lower ileum. One had been operated upon 20 days previously for "spermoblastoma" and adhesions had caused a partial obstruction. One had had an entero-anastomosis five years previously. In all three cases the bolus which had accumulated proximal to the obstruction consisted of mushrooms. With the exception of these three cases, the obstruction was in the terminal ileum. In only one case was the correct diagnosis made before operation. A boy, in a contest, had eaten a huge quantity of unseeded cherries. In practically every case the symptoms were simply those of a partial or complete obstruction of the small bowel.

The ingestion without proper mastication, in short, literally gulping down, a huge quantity of food rich in fiber, throws into the intestine more than it can handle. It is not surprising that under such conditions impaction occurs. It is remarkable that it does not occur more frequently.

In the 28 cases there were 13 deaths, and abstracts of two cases did not give the result. Therefore there was a known mortality of 46 per cent and possibly higher.

CASE REPORT

Mrs. J. M., aged 26, had been married five years and had one child, aged four. Her menstruation had always been normal. No leukorrhea, no urinary symptoms, no previous operation, bowels regular.

Present Illness.—Three days before admission, patient ate a large amount of collards for dinner and another large amount for supper. At 9 P.M. she took a dose of castor oil, not because of abdominal symptoms, but because she had coryza and a severe headache. At 2 A.M. she was awakened with severe abdominal pains which were intermittent and described by her as simulating "labor pains in region of naval." She began vomiting, which was repeated intermittently until admission to the hospital three days later. Several hypodermics of morphia were given during the interim. The abdominal pains, however, continued to become more severe. Her bowels had moved fairly well the day previous to admission and an enema had given good results. She was examined 54 hours after the onset of symptoms.

Physical Examination.—Patient does not look to be desperately ill, but is obviously suffering from rather severe abdominal pains. Heart, lungs and extremities normal. Abdomen is uniformly and moderately distended. There is no marked tenderness anywhere in the abdomen, nor is there any great degree of muscle spasm or rigidity. Over the right lower quadrant there is slight but definite tenderness on moderate pressure. Also the lower right rectus is somewhat more rigid than the rest of the abdomen. Pelvic examination shows the uterus and adnexa to be normal and no tenderness in the vaginal vault. The leukocyte count was 10,200, 79 per cent polymorphonuclears. The urine was negative except for a trace of albumin.

Preoperative Diagnosis.—Subacute appendicitis with obstructive symptoms.

Operation.—Under ether a right rectus incision was made. The appendix was adherent and was removed. In the ileum about one-half inch from the cecum and extending proximally about eight inches could be felt an elongated, sausage shaped, soft, putty-like mass, about the size of a banana. A rubber clamp was placed on the ileum proximal to the mass and the ileum opened with the cautery. The semisolid mass was removed and found to be composed almost entirely of undigested collard leaves matted together. The total amount of this mass after removal was estimated to be about the size of a small orange.

An enterostomy was done. Hypodermoclysis was started following the operation. The patient continued to vomit throughout the day, the ejecta having a definite fecal odor.

Nasal suction drainage was established. At the end of 48 hours there was still only a very slight drainage through the enterostomy tube. A small amount of salt solution was then forced through the enterostomy tube after which fecal drainage became freer, followed by the expulsion of several pieces of collard.

After that the distention disappeared, the patient making an uneventful recovery, leaving the hospital three weeks from the time of the operation with the fecal fistula almost closed.

Discussion.—Dr. J. A. Crisler, Jr. (Memphis, Tenn.).—In connection with food impaction of the small intestine, I wish to report briefly the case of a man, aged 75, who was operated upon 11 months ago for symptoms of partial intestinal obstruction. A mass resembling a gallstone, but actually a fecal mass, was removed from the jejunum. Nothing more was found and an enterostomy was performed. The patient made a complete recovery but six months later was readmitted to the hospital for acute intestinal obstruction. Operation at this time revealed a segment of small intestine, about eight inches long, edematous, distended, packed with food particles, and feeling not unlike a soggy bean bag. An adhesion of the jejunum to the parietal peritoneum at the site of the previous enterostomy had narrowed the lumen of the intestine sufficiently to hold back this large bolus of food. After freeing the adhesions the food particles were evacuated from the edematous segment of the intestine. The patient again recovered and afterwards told us that he had been eating cantaloup without using his false teeth.

Dr. L. Wallace Frank (Louisville, Ky.).—I wish to report experiences with two similar cases. In neither was there a history of the type of food taken. Both patients had symptoms of partial obstruction. In the first case there had been obstruction for four days. At operation a mass was found in the lower end of the jejunum which was thought to be a gallstone. The gallbladder was examined but showed no evidence of disease. An incision was made in the intestine, the mass was removed and found to be inspissated fecal material containing a good deal of fiber. The patient died four days later from peritonitis.

The second case was seen after the symptoms of obstruction had existed for 24 hours. Inasmuch as the first patient had died of peritonitis, and thinking possibly that the sutures in the intestinal wall had sloughed, I decided to resect the obstructed loop rather than to remove the obstructing material. The obstruction was in the midpart of the jejunum and a resection of six inches of intestine was done followed by end-to-end suture. Examination of the excised loop proved the obstructing material to be inspissated intestinal content, again containing a good deal of fibrous material. Pathologic examination of the intestinal wall revealed thrombosis of all the vessels in it. This finding explains, I think, the cause of death in the first patient, namely,

sloughing in the suture line due to impaired circulation in the intestinal wall. In the future, should we be so fortunate as to see another such case, I certainly intend to perform a resection of the involved loop rather than a simple removal of the obstructing mass with subsequent closure of intestine.

Dr. Robert L. Rhodes (Augusta, Ga.).—I would like to recite an instance of food bolus in the intestine cured by nonoperative measures. Seven years ago, following the visit to our community of an advocate of certain dietary measures, including the use of bran, the patient in question thought that if a little was good, a lot would be better, and consumed an entire box of bran within 24 hours. She had vomiting and abdominal pain, and when I saw her there was a mass which could be felt over the region of the terminal ileum. Her bowels had not moved. Her past history was negative. Under hot turpentine stupes, stopping everything by mouth and giving repeated enemata, with an effort to retain the fluid as long as possible, and judicious massage, by a careful nurse, kneading the mass, she succeeded in disseminating it so that the patient was relieved after about 24 hours.

Dr. Waller O. Bullock (Lexington, Ky.).—I would like to add one case to this series, although I hesitate because in some respects it is a source of mortification, but in others it was fortunate. The patient was suffering from a gastric ulcer and we were in doubt as to whether carcinoma was present or not. At operation I dissected the pylorus away from the pancreas. A specimen was examined pathologically at the time and pronounced benign. I therefore performed a pyloric resection and made an anastomosis with the duodenum by an end-to-end procedure; the patient recovered and went home. Three months later he returned with signs and symptoms of acute intestinal obstruction. We operated immediately and found a mass in the jejunum. The jejunum was opened and the mass extracted. It turned out to be a phytobezoar composed of persimmon seeds. A postfacto history revealed the fact that he had eaten persimmons about a year before. He made a complete recovery following operation.

SHOULDER AND CLAVICULAR PAIN IN APPENDICITIS

EDWARD VERNON MASTIN, M.D., M.S.

ST. LOUIS, MO.

FROM THE SURGICAL DEPARTMENT, ST. LOUIS UNIVERSITY SCHOOL OF MEDICINE, ST. LOUIS, MO.

IN 1923, my attention was first called to a case of appendicitis in which pain was referred to the region of the shoulder and clavicle. The second case was seen 11 years later. Recently 485 questionnaires were sent out relative to this subject. Three hundred fifty-one replies were received, among which three instances were reported. A complete review of American and foreign literature from 1920 to 1935 revealed six additional cases, bringing the total up to 11.

Both Siegmund Erben[1] and Jonas[2] mention the fact that they have observed right shoulder pain in appendicitis, but they do not give any case records. Zachary Cope[3] reports two cases of appendicitis where the pain was referred to the right shoulder and clavicle about 15 hours after the onset of symptoms. At operation the appendix was found to have ruptured and there was beginning peritonitis. He states that it is seldom that phrenic shoulder pain is a symptom in uncomplicated appendicitis. He has been able to find references to only two such cases in the continental literature. In a review of 344 cases of appendicitis, David Hilton[4] reports one case where the pain was referred to the region of the right shoulder.

In 1929, Sir Bruce-Porter[5] reports the history of a man, aged 51, who consulted him on account of pain above the right nipple area, of five years' duration. At times this pain was quite severe. During the course of his examination, pressure was made over the region of the appendix, and the patient immediately placed his hand above his right nipple and cried out, "Why, that is my pain," and each repetition of the pressure over the appendix produced an exacerbation of the pain above the nipple. At operation, the appendix was found lying in the pelvis and showed evidence of both acute and chronic inflammation. When the patient recovered from the anesthetic his pain above the nipple had disappeared, and did not return.

Sir Bruce-Porter reports a second case of a young officer who consulted him on account of pain above the left nipple, which the patient attributed to heart disease. Further examination with investigation of his digestive tract revealed chronic appendicitis, and removal of his appendix cured his precordial pain. Irving Gray[6] reports a case of chronic appendicitis with pain referred to the left shoulder. After removal of his appendix, there was no recurrence of his shoulder pain. Fred W. Bailey[7] reports a case of appendicitis with pain referred to the right shoulder and clavicle. The appendix was acutely inflamed, but was free from adhesions. The patient was relieved of the shoulder pain following operation. Francis Reder[8] reports two cases

of appendicitis where the pain was referred to the left supraclavicular and acromial regions and was relieved following appendectomy.

CASE REPORTS

Case 1.—Miss H. S., aged 16, was admitted to St. Luke's Hospital August 2, 1923. On the day before her admission, she was suddenly seized with a sharp pain in the

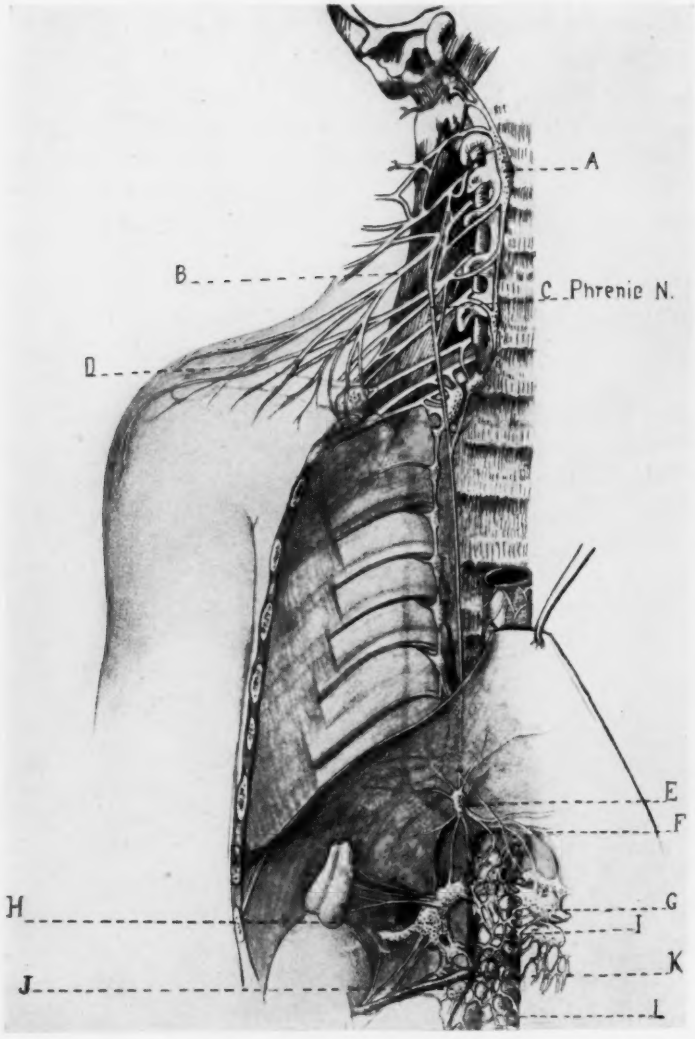


FIG. 1.—Drawing showing the cervical plexus and the cutaneous distribution of the fourth and fifth cervical nerves, also the origin and distribution of the phrenic nerve. (A) Superior cervical sympathetic ganglion. (B) Fourth cervical nerve. (C) Phrenic nerve. (D) Fifth Cervical nerve. (E) Diaphragmatic ganglion. (F) Diaphragmatic plexus. (G) Left semilunar ganglion. (H) Right semilunar ganglion. (I) Celiac plexus. (J) Superior mesenteric ganglion. (K) Superior mesenteric plexus. (L) Aortic plexus.

epigastrium which was referred to the region of her right clavicle, and extended over the right shoulder and down the side of her arm. This was associated with nausea and vomiting. The shoulder pain was increased when she was flat in bed and was partially

relieved when her legs were flexed. She had no pain in the right iliac fossa, but had definite tenderness over the region of her appendix, pressure over which caused severe pain over the region of the clavicle and shoulder. There was beginning rigidity of the right rectus muscle, and hyperesthesia of the right shoulder. The white count was 18,600, with 89 per cent polymorphonuclears. The urine was negative. Examination of her heart and lungs was normal.

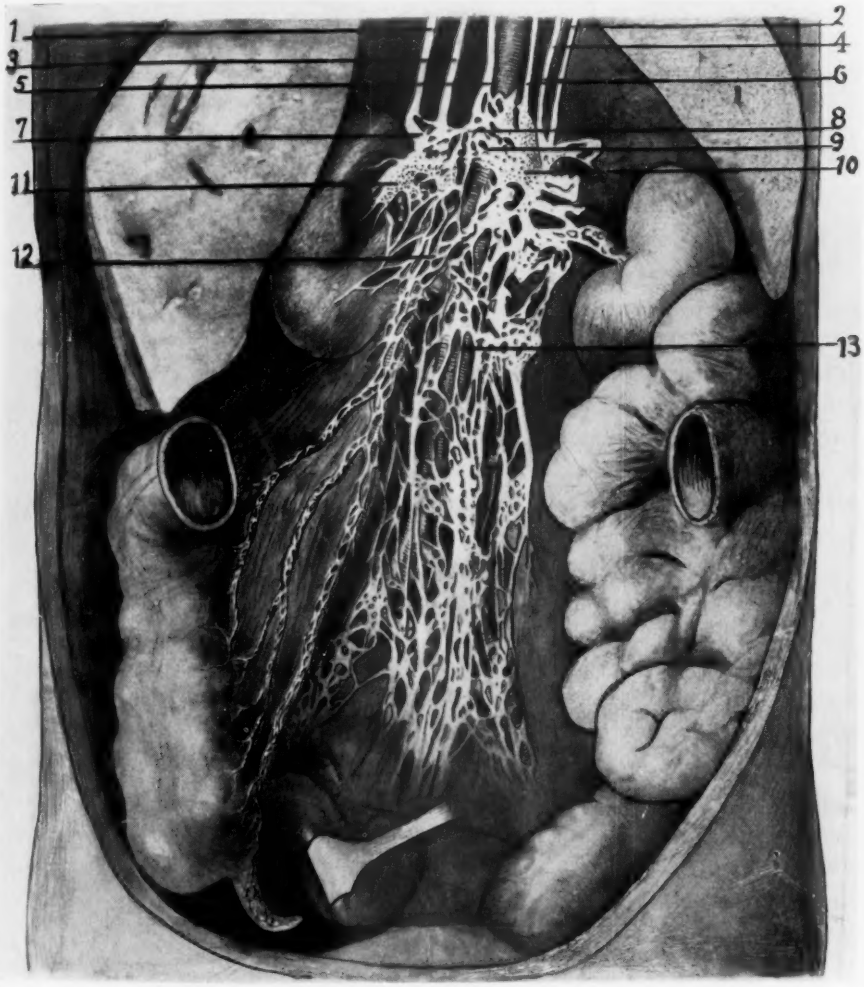


FIG. 2.—Drawing showing the nerve supply to the appendix and the communication between the superior mesenteric plexus and the phrenic nerve. (1) Right phrenic nerve. (2) Left phrenic nerve. (3) Right great splanchnic nerve. (4) Left great splanchnic nerve. (5) Right vagus nerve. (6) Left vagus nerve. (7) Diaphragmatic ganglion. (8) Diaphragmatic plexus. (9) Celiac plexus. (10) Left semilunar ganglion. (11) Right semilunar ganglion. (12) Superior mesenteric plexus. (13) Aortic plexus.

Operation revealed an acutely inflamed appendix that was bound down by dense adhesions in the right iliac fossa. When the patient regained consciousness, she stated that she no longer had any pain in the region of her shoulder.

Case 2.—Mrs. J. B., aged 34, was admitted to St. Luke's Hospital March 19, 1934. While she was eating her dinner she was suddenly seized with a severe pain above the right clavicle which radiated upward toward her neck, and over the side of her shoulder

and down the side of the arm. This pain was associated with nausea and vomiting. The pain in her shoulder continued and she vomited several times during the evening. The nausea subsided that night, but the pain remained constant. A diagnosis of cervical neuralgia was made and her shoulder was baked with an infra-red lamp, but without relief.

Toward the evening of the second day, she had a rather severe pain in the epigastrium with radiation of pain across her chest and over the right shoulder. Dr. L. P. Gay sent her to the hospital with a diagnosis of an acute surgical abdomen. Examination showed definite rigidity of the entire right side of the abdomen and very acute tenderness over the appendiceal region. Whenever pressure was made over the appendix, the pain in her shoulder was increased. There was hyperesthesia about the distribution of the fourth and fifth cervical nerves. The heart and lungs were normal. The urine was normal except for a few white blood cells. White blood count was 21,400, with 92 per cent polymorphonuclears. Temperature, 99.8° F.; pulse, 104.

Operation revealed a very acutely inflamed appendix that was lying free, just above the brim of the pelvis. As soon as the patient regained consciousness, she stated that the pain across her chest and about the region of her shoulder had been relieved.

Relief of the pain in the upper chest and shoulder region following appendectomy indicates that the pain was caused by inflammation of the appendix, consequently it was referred pain. Since the area to which the pain was referred is supplied by the third, fourth and fifth cervical nerves, the afferent impulses arising at the site of the inflammation must have entered the spinal cord in the corresponding cervical segments. The only nerve connected with this segment of the spinal cord, which includes afferent fibers that reach the abdomen, is the phrenic. Consequently these must be regarded as cases in which the pain was referred via the phrenic nerve.

The subject of referred pain has always been an interesting one. It has been definitely proven by the work of Head,[9] Ross[10] and others, that the cutaneous distribution of pain referred from a visceral organ falls within the area of distribution of the nerves, which are connected with the same segments of the spinal cord, as those through which the impulses travel from the diseased viscus. In the cases in question, the pain was in cutaneous areas that are supplied by nerves which arise from the same spinal segments from which the phrenic nerve takes its origin. The supraclavicular and acromial branches of the cervical plexus supply this area, and it will be noted that the phrenic is also a branch of this plexus. The pain impulses originating in the appendix must have been conducted by phrenic nerve fibers which traverse the celiac and superior mesenteric plexuses.

The phrenic nerve is an internal muscular branch of the cervical plexus, arising very largely from the fourth cervical nerve, and receiving twigs from the third cervical and occasionally from the fifth. It contains both motor and sensory fibers, in the proportion of about 2 to 1.

It communicates directly in the lower part of the neck with either the inferior or middle cervical sympathetic ganglion, and just above the diaphragm it receives a twig from the sympathetic cord. The superior cervical sympathetic ganglion sends gray rami communicantes to the first, second, third and fourth cervical nerves and hence to the phrenic; and the ganglion of

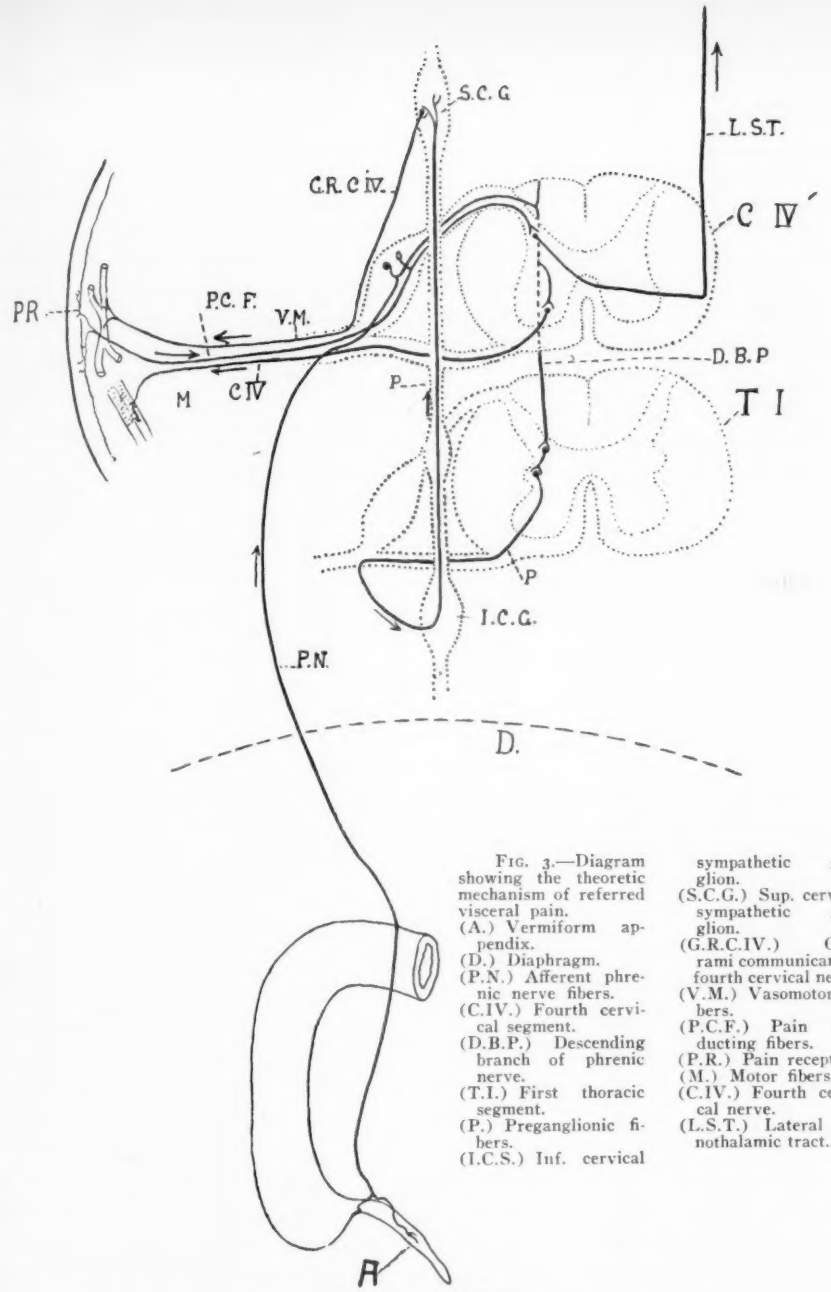


FIG. 3.—Diagram showing the theoretic mechanism of referred visceral pain.
(A.) Vermiform appendix.
(D.) Diaphragm.
(P.N.) Afferent phrenic nerve fibers.
(C.IV.) Fourth cervical segment.
(D.B.P.) Descending branch of phrenic nerve.
(T.I.) First thoracic segment.
(P.) Preganglionic fibers.
(I.C.S.) Inf. cervical sympathetic ganglion.
(S.C.G.) Sup. cervical sympathetic ganglion.
(G.R.C.IV.) Gray rami communicantes, fourth cervical nerve.
(V.M.) Vasomotor fibers.
(P.C.F.) Pain conducting fibers.
(P.R.) Pain receptors.
(M.) Motor fibers.
(C.IV.) Fourth cervical nerve.
(L.S.T.) Lateral spinothalamic tract.

EXPLANATION OF FIG. 3.

The afferent pain impulses originate in the appendix (in these cases), and travel upward over the phrenic fibers through the fourth cervical nerve and make reflex connections in the spinal cord with ordinary motor neurones, and also with preganglionic neurones. Since there are no preganglionic neurones at the fourth cervical level, the impulse must travel downward through the spinal cord over the descending branch of the phrenic nerve to the level of the first thoracic segment; where reflex connections are established with preganglionic neurones. The impulse then continues through the preganglionic fibers and enters the inferior cervical sympathetic ganglion and continues upward to the superior cervical sympathetic ganglion, where synaptic connections are effected with sympathetic motor neurones, and the impulse continues peripherally over the gray rami communicantes of the fourth cervical nerve. Whenever reflexes pass through these sympathetic neurones, they produce vasoconstriction, which results in the liberation of a chemical substance which stimulates the pain receptors in the skin. These impulses are then conducted centrally over the pain conducting fibers of the fourth cervical nerve and are transmitted to the neurones in the gray matter of the cord which join with fibers that ascend in the lateral spinothalamic tract to consciousness; which is the ordinary ascending conductive pathway for impulses of pain.

the trunk of the vagus is sometimes connected by means of a tiny nerve with the loop between the first and second cervical nerves.

The branches of the phrenic nerve are the pleural, pericardial and terminal. According to Gray's[11] anatomy, branches have been described as passing directly to the peritoneum. The terminal branches differ to some extent on the two sides. The right phrenic is divided into two branches, an anterior and a posterior. The anterior branch sends five or six fine twigs to the diaphragmatic musculature. The posterior branch passes through the central tendon of the diaphragm and gives off muscular branches and is continued as the phrenico-abdominal branch.

The phrenico-abdominal branch breaks up, and some of its fibers enter the diaphragmatic ganglion, while others unite with branches of the celiac plexus to form the diaphragmatic plexus. This connection of the phrenic nerve with the celiac plexus affords a pathway through which afferent fibers of the phrenic may, in some cases, reach the superior mesenteric plexus to be distributed to the intestine including the cecum and appendix, through the nerves accompanying the superior mesenteric artery and its branches.

The left phrenic nerve pursues a similar course, but divides into three branches—(a) anterior, (b) lateral, and (c) posterior. The anterior and lateral branches supply the diaphragm. The posterior branch also gives a branch to the diaphragm and is then continued as the phrenico-abdominal branch, sending branches to the left semilunar ganglion, and celiac plexus.

There is a direct communication between the diaphragmatic plexus, the semilunar ganglion, the celiac plexus, and the superior mesenteric plexus. The superior mesenteric plexus is continuous above with the celiac plexus and with the aortic plexus below. The fibers are derived from the semilunar ganglion, the celiac plexus and the right vagus nerve and are supplied to the same portion of the intestinal tract that receives its blood supply from the superior mesenteric artery. Since both the phrenic nerve and the superior mesenteric plexus communicate with the semilunar ganglion and the celiac plexus, and since both receive filaments from the right vagus nerve, it seems only fair to assume that there is a communication between the phrenic nerve and the branches of the superior mesenteric plexus that supply the appendix.

A simple anatomic explanation for clavicular or shoulder pain in appendicitis has been offered, but this is not the whole explanation for such an unusual referred pain, and there must be some other factor or factors present, such as anomalous direct communication between the superior mesenteric plexus and the phrenic nerve, or an anomalous connection through the sympathetics, the splanchnics or the vagus nerve, otherwise this symptom complex would occur more frequently.

CONCLUSIONS

(1) Pain that is referred to the shoulder and clavicular regions in appendicitis is a rare symptom.

(2) This pain ceases immediately after removal of the appendix.

(3) Pain in the shoulder and clavicular regions due to appendicitis can be explained only as referred pain resulting from impulses arising from the appendix and conducted through the phrenic nerve.

(4) A possible explanation of this symptom complex is offered by an anatomic diagram, showing a communication between the phrenic nerve and the terminal branches of the superior mesenteric plexus.

(5) Distribution of phrenic fibers to the appendix must be regarded as anomalous, otherwise this symptom complex would occur more frequently.

REFERENCES

[1] Erben, S.: Diagnostic Significance of Pain. Wien. med. Wchnschr., vol. 83, pp. 1110–1113, 1933.

[2] Jonas, M.: Quoted by Brohee, G.

[3] Cope, Z.: Clinical Study of Phrenic Shoulder-pain with Special Bearing on Diagnosis of Acute Abdominal Disease. Brit. Jour. Surg., vol. 10, pp. 192–201, 1922. The Significance of Subclavicular Pain in Lesions Near the Diaphragm. Brit. Med. Jour., vol. 1, pp. 383–384, 1930. Clinical Significance of Shoulder Pain in Lesions of Upper Abdomen. Proc. Roy. Soc. Med. (Clin. Sect.), vol. 14, p. 11, 1921.

[4] Hilton, David C.: Appendicitis: A Review of 344 Cases Taken from the Records of the Bryan Memorial Hospital of Lincoln, Nebraska, from June 7, 1926, to June 7, 1930. Internat. Clin., vol. 4, pp. 244–264, 1932.

[5] Bruce-Porter, B.: Unusual Referred Pain in Case of Chronic Appendicitis. Lancet, vol. 2, p. 495, 1924.

[6] Gray, I.: Left Shoulder Pain of Phrenic Origin, Reflex Symptom in Chronic Appendicitis; with Report of Three Cases. Am. Jour. Med. Sci., vol. 170, pp. 894–899, 1925.

[7] Bailey, Fred W.: Personal communication.

[8] Reder, Francis: Personal communication.

[9] Head, Henry: On Disturbances of Sensation with Especial Reference to the Pain of Visceral Disease. Brain, vol. 16, p. 1, 1893; vol. 17, p. 339, 1894; vol. 19, p. 153, 1896. Also Certain Aspects of Pain. Brit. Med. Jour., vol. 1, pp. 1–5 (illus.), 1922.

[10] Ross, J.: On the Segmental Distributions of Sensory Disorders. Brain, vol. 10, p. 33, 1888.

[11] Gray, Henry: Gray's Anatomy. Lea and Febiger, Philadelphia and New York.

Discussion.—Dr. Hubert A. Royster (Raleigh, N. C.)—In 1927, I made a collection of signs of appendicitis, 14 in number. I arranged them alphabetically, and am glad to add another sign under the letter "M." This is Mastin's sign. Some of the signs are purely reflex. Others are toxic in origin, proving that the appendix is the focus of infection. The reflex is seen in areas in which we can reproduce the pain, for example, in the epigastrium, at which site the patients complain in acute attacks. This sign of Doctor Mastin's is in the same category. The reflexes are centered in the midbrain and the subconscious feeling is produced accordingly. In the other type of acute pain due to absorption, perhaps, in disease of the appendix, there are headache, trigeminal neuralgia, sciatica, and so on. These signs are especially valuable, it seems to me, in the intervals of so called recurrent or relapsing cases. They can be elicited in various ways. Rolling to the left side will reproduce the pain in the appendix. I think distinction should be made between the neurogenic cases and those of toxic origin. The toxic origin is so important that I have called attention to the three T's, teeth, tonsils and "tummy," the latter meaning foci of disease in the abdominal cavity, which are of equal importance with the first two.

Dr. Joe V. Meigs (Boston, Mass.)—We have had a few cases of pain in the shoulder following hemorrhage from a ruptured follicle or corpus luteum of the ovary. I wonder if this pain could not have been reproduced by the method of nerve reference described by Doctor Mastin.

Dr. Guy L. Hunner (Baltimore, Md.)—I think we should bear in mind that renal pain is sometimes located in the chest and we must take it into consideration in diagnosing this group that Doctor Mastin has spoken to us about. Usually if the shoulder or scapular pain is of very acute character, of course we have to differentiate it more frequently from gallbladder pain. If it is of more chronic and of less acute character we often think of heart trouble if it is on the left side, or of tuberculosis if it happens to be on one or both sides, particularly if there is a little increase of temperature, as there is in some of the renal absorption cases. The way in which it might be mistaken for the pain of appendicitis is that on palpation of the tender ureter as it crosses the pelvic brim, unless you have both the appendix and the ureter in mind, you may conclude that it is the appendix. I have had three renal cases who had pain referred to the chest and had various periods of confinement in tuberculosis sanatoriums, although there was no positive evidence of tuberculosis. They were sent on the strength of the chest pain and a low degree of fever which was more or less continuous. These cases clear up when relieved of back pressure on the kidney.

Dr. E. V. Mastin (closing).—In answer to Doctor Meigs' question, I can see no reason why the pain mentioned could not be traced over a similar arc. The afferent fibers would have to travel through the hypogastric, aortic and semilunar plexuses, through the phrenic nerve, in the same way which I have described.

One reason that prompted me to present this paper was the hope that physicians would be on the lookout for this symptom complex, as I am sure there must be many more cases in which it is present, but overlooked.

Copps and Coleman, in 1922, reported the results of their experiments in cases where a trocar was inserted through the abdominal wall for relief of ascites. They next passed a curved wire and stimulated various parts of the peritoneum and the under surface of the diaphragm. When the diaphragm was stimulated, and hence the branches of the phrenic nerve, the pain was referred to the shoulder tip, but when the other areas of the peritoneum were stimulated, the pain was localized very closely to the point of stimulation. These results have been duplicated by other observers.

Morley has done a great deal of interesting work on referred visceral pain and has shown that when the terminal branches of the fourth and fifth cervical nerves are anesthetized, the shoulder tip pain does not occur.

SERIOUS COMPLICATIONS FOLLOWING NEGLECTED CHOLELITHIASIS

FRED W. BAILEY, M.D.
ST. LOUIS, MO.

BUT one phase of hepatic disease will be considered, *i.e.*, cholelithiasis, and my observations will be limited to the consideration of the serious complications of this condition, and suggestions for their prevention. Problems of diagnosis and treatment are necessarily omitted, as the entire object is to emphasize two pertinent points: (1) that symptomless gallbladder pathology is a rarity; (2) the infected gallbladder with gallstones is never cured by duodenal drainage nor by medical therapy. Its pathology is progressive and potentially dangerous to life and health, and merits the recognition of primary symptoms, early diagnosis and preventive surgery. To approach control of serious surgical disease, we cannot ignore the demand of early recognition. This will not be practically available without the interested cooperation of the patient, physician and surgeon.

Although it is not within the scope of this presentation to discuss the fundamentals of gall tract disease, its symptomatology, pathology, or treatment, it would be discourteous to write even the briefest monograph on this subject, or any part of one, without giving full credit to the pioneers in gall tract study such as Entmuller, Malpighi, Van der Wiel, Muller and Jean Louis Petit, and to the masters of the nineteenth century, among whom were Langenbach, Marion-Sims, Kocher, Tait and Keen.

At the opening years of the present century, our knowledge of gallbladder disease rested chiefly upon rather crude assumptions. As pointed out by Judd, the gallstone was considered the "bandit" of gallbladder disease, but conservation of the gallbladder, however deeply infected, was deemed essential. Within 35 years, through the masterly surgery, persistent experimentation, pathologic and bacterial studies of Mayo-Robson, Deaver, Moynihan, W. J. Mayo, C. H. Mayo, Rowntree, Mann, Rosenow and many others, we are now aware that an infected gallbladder is a menace to health; does not spontaneously heal without residual pathology; promotes and fosters formation of stones, pericystic adhesions and liver dysfunction; and may be removed without detriment to health. As stressed so earnestly by Judd and others, the need of continuous cooperation between the laboratorian, the internist and the surgeon is paramount if we hope to control disease. By the brilliant work of Mann, Graham, Rowntree, Van den Bergh and others, we are approaching a working knowledge in early diagnosis and the effect of disordered liver function upon the blood stream, pancreas, stomach, intestines and metabolic activities. A satisfactory training in pathology and ther-

apy is best secured by a thorough knowledge of the physiology of the organs involved. Through these pioneers and their co-workers, in their various fields of research, we have been furnished a broader knowledge of the function and physiology of the liver and its contiguous organs.

It is customary to rate our estimate of the gravity of a disease by the number of deaths which occur. Should we not instead predicate such an estimate upon the length and degree of morbidity, with its violent strain upon vital functions, economic distress, pain, discomfort and fear, rather than upon final and complete dissolution? Defeat of death by operation in a stage which assuredly leaves a residue of permanent disability dare not challenge comparison with the result following an early diagnosis and operation before definite damage is sustained, particularly in a disease which we are persuaded is surgical from the period of the initial gallbladder infection. Each case so conducted, before hepatic function is disturbed, obviously banishes most of the morbidity which surely follows neglect, such as cholangeitis, chronic hepatitis, biliary obstruction, perforation, abscess, peritonitis, crippling adhesions, pancreatic disease and possibly cancer.

At no age in life are gallstones harmless, and a neglected case is always a menace to the well being of the carrier. Two hundred cases were reviewed, the ages ranging from 28 to 86, with symptoms of gallbladder disease present and active from five to 60 years. Periods of morbidity were variable and the complications were uniformly serious (Table I).

TABLE I

COMPLICATIONS ENCOUNTERED IN TWENTY-EIGHT CASES OF CHOLELITHIASIS

Hydrops	2
Hour-glass gallbladder	2
Gallbladder completely impacted	3
Single stone, cystic duct impaction with obstruction	5
Gallbladder stones and cystic duct impaction with obstruction	13
Gangrenous cholecystitis with gallstones	6
Perforation with abscess	12
Perforation into liver abscess containing stones	3
Common duct stones with obstruction	3
Common duct occlusion (stones, abscess, edema, Ca.)	9
Cancer involving cystic duct area	2
Mortality (28 cases reviewed; 2 Ca. lived 6 months and 1½ years respectively. One died 3rd postoperative day, cardiorenal)	
One case only, free of adhesions	

Each of these patients was of necessity subjected to a serious operation, long tedious hospitalization and subsequent morbidity out of all reasonable proportion to that which would have attended an earlier diagnosis and lowered surgical risk during the period of safety.

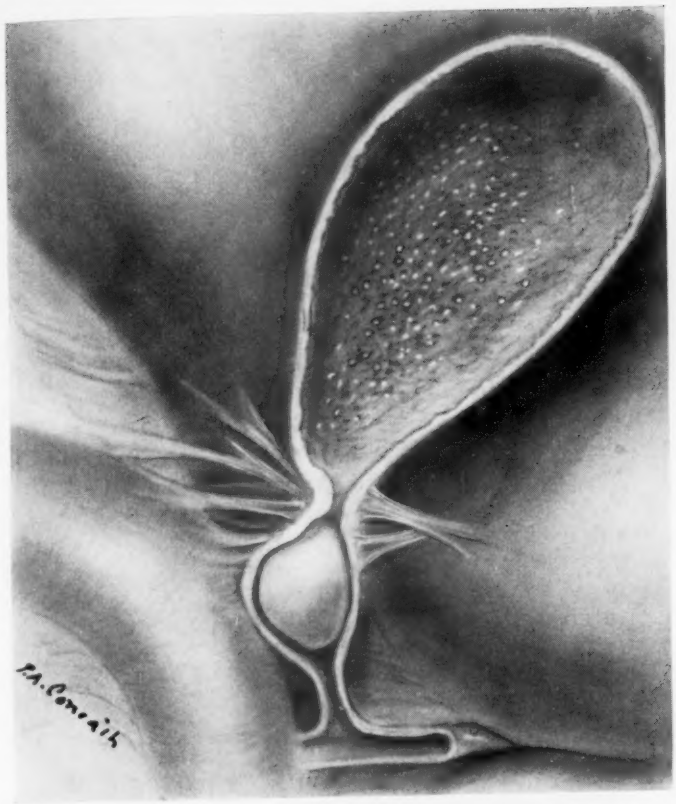


FIG. 1 (Case 6).—Hour-glass gallbladder. Impacted cystic duct stone.

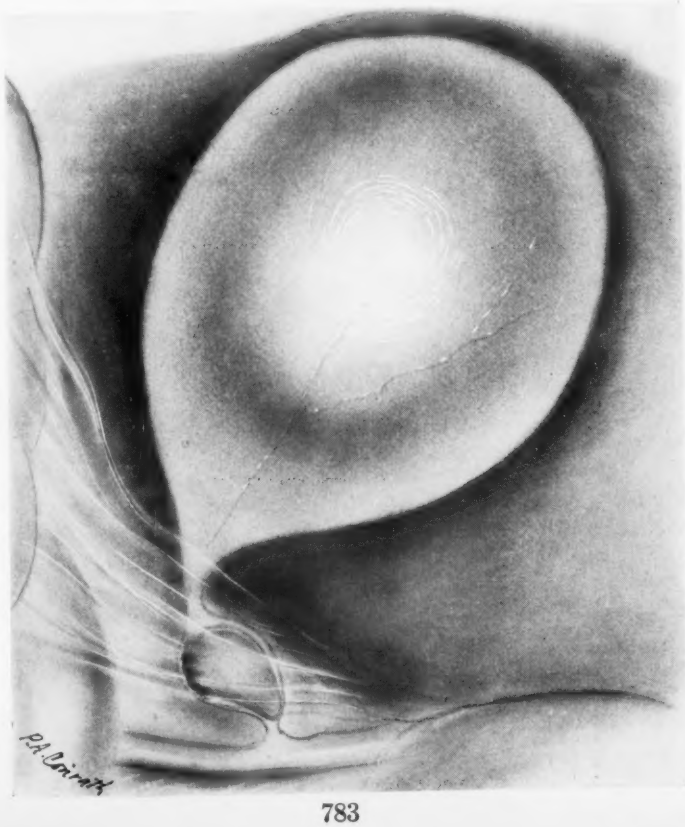


FIG. 2 (Case 7).—Impacted cystic stone. Beginning gangrene.

ABSTRACTED HISTORIES OF TWENTY-EIGHT CASES WHICH PRESENTED SERIOUS COMPLICATIONS

Case 1.—Mrs. L. F., aged 60. Had been semi-invalid for nearly 30 years; continuous epigastric distress. A typical psychoneurotic. Had four previous operations. Iodeikon test revealed diseased gallbladder with stones.

Operation revealed liver margin adherent to diaphragm, numerous adhesions with duodenum entirely covering gallbladder. Many gallstones removed by cholecystectomy. Entirely relieved of distress and nervous symptoms.

Case 2.—Mrs. H. W., aged 48. First attack of upper abdominal pain 20 years ago, intermittent attacks of indigestion since. Final attack progressive. Patient had developed severe cardiovascular-renal disease with hypertension.

Operation revealed two large stones completely filling inactive gallbladder. Marked improvement followed cholecystectomy.

Case 3.—J. W. H., aged 51. Suffered acute attack of epigastric colic 27 years ago, with classic symptoms indicating gallbladder infection. Practiced medicine for 25 years with frequent attacks of upper abdominal pain, nausea and vomiting, jaundice not marked except for few days following each attack.

Operation revealed small contracted gallbladder, fixed to transverse colon and duodenum, containing single large impacted gallstone.

Case 4.—Mrs. R. L., aged 30. Attacks of indigestion, with pain in gallbladder region for several years. Final attack progressive.

Operation revealed gallbladder filled with stones, attached to and compressed by a tumor lying between the cystic area and the duodenum. The tumor peeled out readily, consisting of pancreatic tissues, non-malignant. Numerous adhesions between hepatic flexure and duodenum and gallbladder.

Case 5.—A. S., aged 55. Indigestion with nausea and dull pain in gallbladder region for several years. Treated continuously for indigestion.

Operation revealed infected gallbladder with cystic duct firmly impacted by large mulberry stone. Numerous adhesions.

Case 6.—Mrs. A. H. L., aged 32. Under treatment for eight years for peptic ulcer. Appendectomy nine years ago. Frequent attacks of epigastric pain, with vomiting and flatulence. Not responding to medical treatment.

Operation revealed hour-glass contraction distal to ampula of gallbladder, which contained an impacted stone. Numerous fibrous adhesions to duodenum and liver.

Case 7.—Mrs. R. M., aged 30. Treated for indigestion for over five years. Final attack unrelieved.

Operation revealed huge hydrops of gallbladder with single impacted stone of cystic duct, which showed area of beginning gangrene. Cystic duct distal to stone completely occluded.

Case 8.—Mrs. N. H., aged 55. Had suffered intermittent attacks of epigastric pain over 25 years, with slight icterus, which faded rapidly. Recent attack progressive.

Operation revealed huge gallbladder filled with faceted stones, with one large stone obstructing cystic duct compressing the common duct. No pericystic adhesions.

Case 9.—Mrs. R. M., aged 51. Gastric symptoms for 20 years. Frequent attacks of epigastric pain with vomiting. Previous gallbladder drainage for acute empyema of gallbladder. This attack progressive.

Operation revealed huge gallbladder, completely filled with disintegrated stones, mucosa necrotic, stone impacted in cystic duct, with numerous adhesions.

Case 10.—A. F., aged 36. Several acute attacks of gallstone colic within the last few years. Treated for indigestion. Final attack persistent.

Operation revealed many adhesions covering the gallbladder completely, filled with

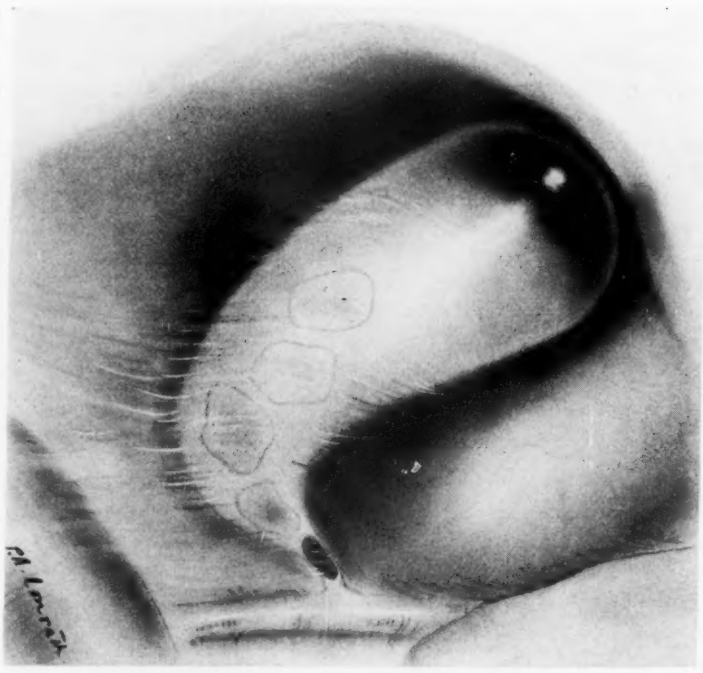


FIG. 3 (Case 15).—Many gallstones, impacted cystic duct. Fundus gangrenous, not ruptured.

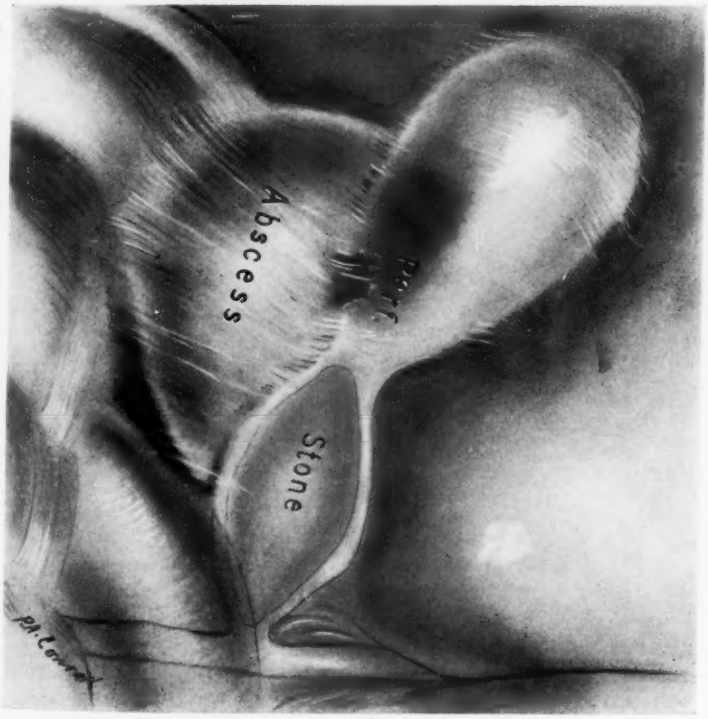


FIG. 4 (Case 16).—Huge impacted cystic stone. Empyema. Gangrenous perforation. Abscess.

stones, one mulberry stone impacted in cystic duct with perforation leading to post-duodenal abscess.

Case 11.—Mrs. N. A., aged 38. Long standing indigestion with intermittent pain in right upper quadrant, accompanied by slight icterus. Present attack subacute but progressive.

Operation revealed hour-glass constriction about middle portion of gallbladder with the cystic portion filled with impacted stones. Common duct compressed and edematous from impacted stone in cystic duct. Multiple adhesions.

Case 12.—Mrs. H. O., aged 31. Several years' history of acute and chronic indigestion. Final attack progressive with jaundice.

Operation revealed a huge infected hydrops of gallbladder with impacted cystic duct stone and ball valve common duct stone, proximal to cystic duct.

Case 13.—Mrs. M. B., aged 86. Persistent jaundice, with severe itching of skin. Pain in upper right quadrant, continuous nausea and vomiting. Demanded relief.

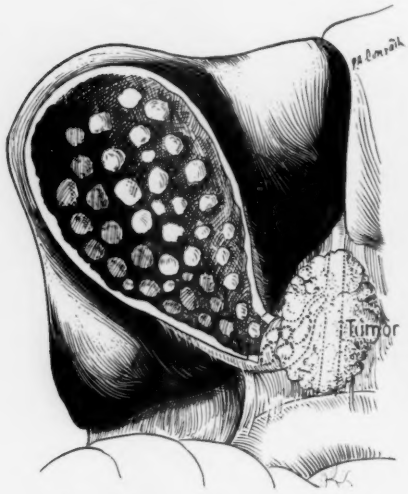


FIG. 5 (Case 18).—Large gallbladder filled with stones. Gangrenous mucosa. Tumor (a) involving cystic and common duct.

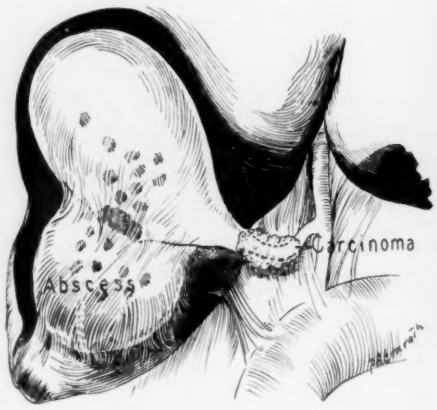


FIG. 6 (Case 19).—Gallbladder ruptured. Abscess containing stones. Carcinoma of cystic duct area.

Operation showed gallbladder filled with stones, single stones obstructing cystic duct and common duct. Removal of stones with drainage of cystic duct and common duct. Symptoms relieved, lived comfortably six years.

Case 14.—Mrs. L. W. M., aged 47. Previous attack of appendicitis; recurrent attacks of epigastric pain for over five years following appendicitis operation. Slight icterus. Final attack acute and progressive.

Operation revealed a large adherent gallbladder filled with stones, with cystic duct obstructed and tip of gallbladder fundus gangrenous, threatening perforation. Two-stage removal.

Case 15.—Mrs. M. V., aged 28. Several years complained of indigestion and biliary colic. Present attack continuous and progressive with symptoms of infection.

Operation revealed greatly enlarged gallbladder, gangrenous at the presenting fundus, filled with large stones, with cystic duct completely obstructed by impacted stone. Numerous pericystic adhesions.

Case 16.—Mrs. Wm. C., aged 36. Gastro-intestinal symptoms for 20 years; fre-

quent attacks of gallbladder colic with transient jaundice. Cholecystostomy 12 years ago. Abdominal exploratory eight and one-half years ago. Present attack progressively acute.

Operation revealed large oval impacted cystic stone with perforation near fundus and abscess lying between gallbladder and transverse colon. Gangrenous at site of perforation.

Case 17.—Mrs. E. H., aged 55. Epigastric pain and indigestion for over 20 years. Developed cardiorenal complex with hypertension. Final attack progressive and acute.

Operation revealed an acute gangrenous, edematous gallbladder, filled with stones, surrounded by mucopurulent abscess walled off by omentum. Drainage under local anesthesia. Cardiorenal death on third postoperative day.

Case 18.—McD., aged 64. For over 20 years, severe attacks of indigestion with almost persistent tenderness over right hypochondrium. Final attack progressive with persistent jaundice.

FIG. 7 (Case 23).—Four distinct abscesses, containing purulent bile. Contracted gallbladder. Many adhesions.

Operation revealed a large gallbladder filled with stones, entire mucosa gangrenous. Tumor (Ca.) involving cystic and common duct area. Drainage only. Fatal issue six months later.

Case 19.—Mrs. B. M., aged 35. Intermittent attacks of gallbladder pain with indigestion, flatulence, abdominal distension, *etc.* Final attack continuous and progressive.

Operation revealed gallbladder filled with stones, with an adjoining abscess containing gallstones; numerous protective adhesions. Condition permitted gallbladder and abscess drainage with a removal of stones only. Subsequent operation refused until recurrent attack one year later. Operation revealed empyema of gallbladder with malignant tumor involving cystic duct area, pancreas considerably enlarged, multiple adhesions. Died one and one-half years later.

Case 20.—C. W., aged 69. Irregular attacks of epigastric pain for 35 years, jaundiced over six months, 28 pounds weight loss. Last attack progressive.

Operation revealed multiple gastrohepatic and colohepatic adhesions, completely

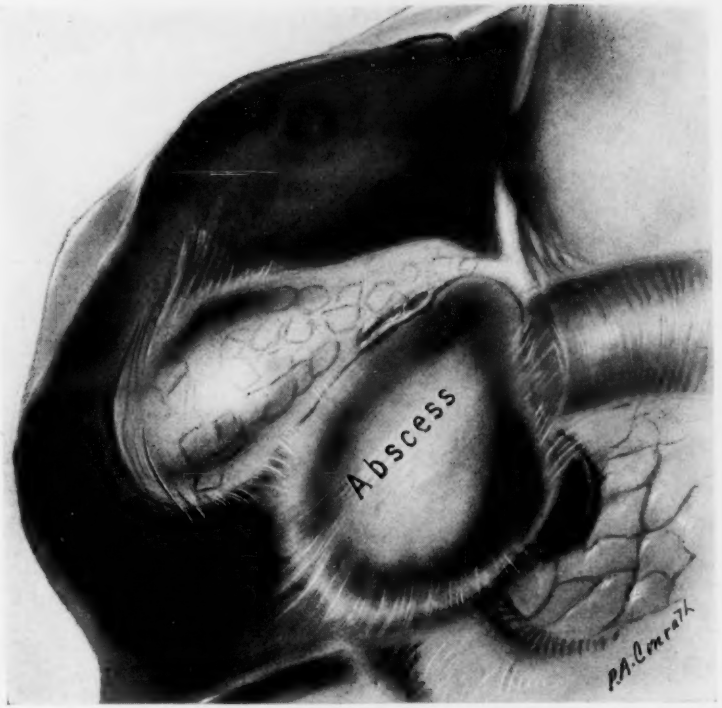


FIG. 8 (Case 24).—Gallbladder filled with stones. Perforation with abscess overlying head of pancreas.

FIG. 9 (Case 25).—Gallbladder and cystic duct filled with stones—impacted. One large stone perforated into abscess.

burying small gallbladder which contained granular stones and one impacted cystic stone with perforation and pericystic abscess.

Case 21.—Miss L. O., aged 50. Seven years' upper abdominal discomfort, flatulence and frequent attacks of pain with icterus, lasting one to two months. Persistent jaundice during final attack.

Operation revealed small abscess, covered by duodenal adhesions. Gallbladder filled with stones, cystic duct presenting an impacted stone of large size which had perforated, forming abscess. Multiple adhesions.

Case 22.—Mrs. J. R., aged 52. Recurrent attacks of pain in epigastrium, bloating and belching, with nausea for more than ten years. Final attack severe.

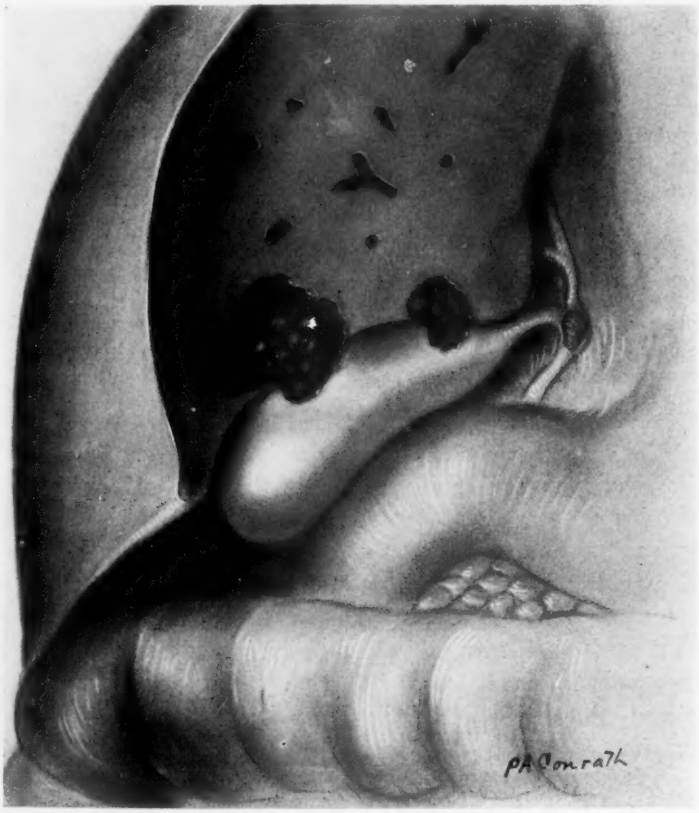


FIG. 10 (Case 28).—Double perforation into liver bed, both abscesses containing stones. Common duct stone impacted.

Operation revealed an old healed perforation near the cystic end of the gallbladder, opposite impacted cystic stone. Entire area between gallbladder and duodenum filled with a granuloma, evidently the result of a preexisting abscess following perforation.

Case 23.—J. W. McI., aged 55. History of infected right kidney, not perinephritic; for several years had mild attacks of indigestion. Six months previous, hospitalized for acute right kidney infection, developed severe pain in lower dorsal spine requiring cast. While in hospital developed symptoms of acute obstructive cholecystitis with perforation and drainage into stomach or duodenum. Rapid recovery for four months; again stricken with severe epigastric pain, temperature and classic symptoms of gallbladder infection.

Operation revealed small contracted gallbladder buried in adhesions. No stones palpable in gallbladder or common duct, duodenum overlying and adherent; there were four distinct abscess cavities, segregated and lying between transverse colon, omentum and liver.

Case 24.—Mrs. F. C., aged 50. Indigestion and nausea with intermittent attacks of epigastric pain for many years. Final attack progressive.

Operation revealed gallbladder completely filled with stones, resting upon a huge abscess overlying duodenum and head of pancreas. Gallbladder perforation had healed.

Case 25.—Mrs. L. S., aged 62. Indigestion for 20 years. Frequent acute attacks of pain with transient jaundice. Final attack progressive.

Operation revealed gallbladder and cystic duct filled with stones, one large stone impacted in perforation with surrounding abscess.

Case 26.—Mrs. E. B., aged 57. Acute painful gallbladder attacks of several years, final attack continuous.

Operation revealed perforation of gallbladder through its bed, into the liver, with abscess formation containing stones.

Case 27.—E. N., aged 51. Intermittent attacks of gallbladder colic for 30 years; several attacks of icterus. Final attack progressive.

Operation revealed acutely infected gallbladder, filled with stones, completely buried in colohepatic fibrous adhesions. One large gallstone had perforated into liver bed near the fundus, surrounded by abscess.

Case 28.—Mrs. J. B., aged 65. Over 20 years of upper abdominal discomfort, treated 15 years for indigestion. Rapid loss of weight preceding final progressive attack. Deeply jaundiced.

Operation revealed large thick walled gallbladder with numerous adhesions from transverse colon and gastrohepatic omentum. Gallbladder filled with stones, two distinct perforations through gallbladder bed into liver substance, with abscess, both cavities containing stones. Single stone impacted at junction of cystic and common duct.

All of the 200 cases reviewed gave positive evidence of varying periods of disabling symptoms; all of them ascribed their early disability to various forms of "dyspepsia," "indigestion," "nervous indigestion" and dietary indiscretions; most of them had been under a physician's care for a prolonged period; the majority had suffered actual biliary colic many times with transient or persistent jaundice, intestinal dysfunction and fading health; among those who were diagnosed, frequent duodenal drainage and bile salts were the prescribed treatment. Several resorted to the assuring care of the stone-dissolving charlatan, the osteopath, chiropractor or to Christian Science. The morbidity of a disease should lessen in proportion to increasing knowledge of the cause and effect and by virtue of improvement in diagnosis and therapeutic control. Such is anticipated in medical progress. This is apparently not true of gall tract disease in general. Our broader knowledge of the physiologic function of the liver and of its susceptibility to disease, our ability to detect pathology early and a constantly improving technic, should arm us with a more effective remedy and yet our end-result in treating hepatic disorder is not comparable to that of any other disease of which infection is the basic cause.

The early concept of the physician's duty was the treatment of disease. We have long known that it is far more commendable and economically advantageous to prevent accident, illness or life-threatening operations when

TABLE II

RÉSUMÉ OF THE TWENTY-EIGHT REPORTED CASES

No.	Age	Years of Indigestion Symptoms	Years of Painful Attacks	Complications	Jaundice	Final Attack
1	60	30	5	Psychoneurotic	None	Interval
2	48	20	20	Cardiorenal	Slight	Prog.
3	51	30	27	Narcotics	W. attk.	Interval
4	30	5	3	Panc. tumor	Slight	Ac. prog.
5	55	20	5		None	Interval
6	32	8	8	App. 9 yrs.	None	Interval
7	30	5	2	Gangrene	Fin. attk.	Ac. prog.
8	55	25	25		Slight	Ac. prog.
9	51	20	7	Perst. vom.	None	Ac. prog.
10	36	10	4		None	Prog.
11	38	12	5	Hour-glass	Slight	Subacute
12	31	10	5		Recurr.	Prog.
13	86	60	15		Persist.	Prog.
14	47	20	5	App. op.	Slight	Ac. prog.
15	28	8	5	Gang. G. B.	None	Ac. prog.
16	36	20	15	G. B. Dr. 12 yrs.	None	Ac. prog.
17	55	20	10	Cardiorenal	None	Ac. prog.
18	64	30	20	Ca. cystic D.	Persist.	Ac. prog.
19	35	10	3	Ca. cystic D.	Slow prog.	Ac. prog.
20	69	35	35	Wt. loss	W. attk.	Ac. prog.
21	50	20	6	Flatulence	Persist.	Ac. prog.
22	52	10	10	Healed perf.	Slight	Ac. prog.
23	55	15	7	G. B. perf. duod.	One attk.	Interval
24	50	20	20	Healed perf.	W. attk.	Ac. prog.
25	62	25	20		Transient	Ac. prog.
26	57	20	10	Scientist	W. attk.	Chr. inf.
27	51	30	30	Psychosis	Sev. attk.	Ac. prog.
28	65	25	20	Wt. loss	W. attk.	Ac. prog.
Average	50	20	12		20 Pos.	5 Int. 23 Ac. prog.

possible. Had we the foresight to expend but a fraction of the time and energy which is applied to eradicating or controlling an active pathology, upon the problem of anticipating the disease, perfecting early diagnosis and applying preventive medicine and surgery during its inception, we could well boast actual progress and more firmly establish our value as custodians of the public health.

It has well been said that the palm of one hand properly placed in the upper right quadrant will cover the source of the major portion of serious abdominal pathology. This indictment may appear unwarranted until we recall that the seemingly innocent presence of gallstones may later account for (a) indigestion mild or severe, but persistent; (b) obstruction of cystic or common duct, with disastrous sequelae, such as hydrops, empyema, gangrene, perforation and abscess; (c) recurrent infections transmitted to the

liver, increasing dysfunction; (d) pain from insignificant soreness to intense colic; (e) jaundice, mild or severe, persistent or recurrent; (f) cholangeitis, pancreatitis, hemorrhagic or cystic and cancer (from 50 per cent to 75 per cent of all cancers of the gall tract and pancreas show the presence of gallstones).

Is gallbladder disease with gallstones a medical problem? Is cholelithiasis ever actually relieved by the spontaneous passing of all resident stones through the natural channels? Is a definitely diseased gallbladder with gallstones ever "cured" spontaneously or by duodenal drainage or other medical therapeutic agents? Assuming a negative answer to these pertinent questions, should we without protest permit this definitely surgical disease to pass from the stage of a safe operative risk, on to the certain development of disastrous sequelae, without fully informing the patient of its potentialities? My experience impels me to classify the infected gallbladder with stones in the same category as the pathologic appendix, the toxic goiter and progressive obstruction as definite surgical problems entitled to prompt surgical attention when diagnosis is certain and before irremedial liver, gall tract and pancreatic damage has been inflicted.

Medical scientists the world over are continually striving to alleviate pain, lessen morbidity and reduce mortality. Whether this be attained by prevention of disease and injury, medical therapy or surgery, preservation of life and health is the objective. Therapeutic discoveries of great value are infrequent. The extension of life and health by surgical effort is largely dependent upon time, technic and surgical judgment. Educational aid, by informing the public of the early symptoms of serious illness and persuading them to accept prompt medical care, is the most pertinent single factor in mortality reduction. Such an effort will miscarry if the family physician fails to adopt a preventive attitude and to cooperate accordingly. A startling reduction in our present mortality rate is possible and feasible, whenever the tremendous value of preventive surgery is recognized and both physician and patient become preventive conscious. Preventive medicine has long been accepted as a most valuable adjunct. Preventive surgery is as yet an embryo and has in no wise kept pace with preventive medicine. A life ultimately saved by a few words of advice or by a safe, early operation is just as valuable as one preserved after a thrilling fight by heroic surgery and definitely lessens the morbidity.

CONCLUSIONS

(1) From a review of 200 gallbladder operations, more than 50 or 25 per cent were seriously complicated, requiring life endangering operations. Twenty-eight are briefly presented.

(2) Infected gallbladder with stone is usually a progressive pathology, gradually merging from a simple to a serious surgical problem.

(3) Persistent symptoms of indigestion, dyspepsia, flatulence or upper abdominal pain justify careful examination and observation. A diseased

gallbladder seldom, if ever, entirely heals, and usually becomes a factory for stones and related complications.

(4) Early or preventive surgery in a frankly surgical disease is a most potent factor for safety, and is quite as essential in gallbladder disease as it is in toxic goiter, appendicitis or chronic intestinal obstruction.

(5) There is urgent need for a closer cooperation of all branches of medical service toward preventive medicine and surgery. Successful prevention will not be realized unless our major and minor medical bodies promote an active educational plan, which will teach the layman to recognize and respect early symptoms of dangerous diseases and to place themselves under competent medical care. Thus will be marshaled an effective enemy of morbidity and mortality.

THE TREATMENT OF AMEBIC ABSCESS OF THE LIVER

HUBERT A. ROYSTER, M.D., H. B. HAYWOOD, M.D.,
AND
W. W. STANFIELD, M.D.
RALEIGH, N. C.

FROM THE MEDICAL AND SURGICAL SERVICES OF REX HOSPITAL, RALEIGH, N. C.

OUR sole purpose in reporting a single case of amebic abscess of the liver is to emphasize the value of its treatment by repeated aspirations in connection with the simultaneous injection of emetine, both intramuscularly and locally, into the abscess cavity. We are not concerned here with the pathogenesis, symptomatology, or diagnosis of amebiasis. Its phenomena have been fully described by clinicians and pathologists in regions where the affection is most prevalent. Its rarity in our immediate geographic area is well known.

CASE REPORT

W. S. H., a farmer, aged 52, was admitted to the Rex Hospital, September 6, 1934, complaining of intense pain in his "right short ribs and back." He dated his illness from two months before admission when he noticed great weakness and drowsiness, but continued to work; three weeks later he "swelled up on his right side," took saline purges for a week, and then went to bed. On September 1, 1934, five days before admission, he had a chill, followed by five others in the course of two days. Since then the pains have persisted, radiating to each side of the chest and back. Further questioning elicited the fact that since 1932 he had had, at intervals, blood in his stools, associated with diarrhea and occasional hematemesis.

Physical examination showed a tall, emaciated man, with drawn, pinched features, sunken eyes and leaking skin. His face expressed anxiety and severe suffering. The chest expansion was poor, but equal on each side, a friction rub was heard over the right sixth interspace anteriorly. There was tenseness on palpation over the right lower chest and upper abdominal quadrant. The liver was enlarged upward to the third rib and downward for three inches below the costal margin. An indistinct sense of "pointing" could be elicited at the eighth interspace laterally. The temperature was 103° F.; pulse, 92; respiration, 22 (Chart I); the blood count showed a leukocytosis of 18,000. The next day the protozoa of *Endameba histolytica* were found in the stools. A diagnosis of amebic abscess of the liver seemed evident. Accordingly, an intramuscular injection of a grain of emetine hydrochloride was immediately given, and orders were left for this to be continued daily. Roentgenologic examination showed the diaphragm pushed evenly upward and it appeared to be under pressure from beneath. The liver enlargment and its increased density were observed (Fig. 1). Meanwhile the patient's condition did not improve; the elevated temperature remained, the pain was increasing, the liver was enlarging and the pulse was becoming more rapid. On the third day, a needle was introduced into the liver through the eighth interspace in the midaxillary line and four ounces of thick, odorless pus were evacuated (Fig. 2). Before removing the needle two grains of emetine were injected through it into the abscess cavity. Encysted forms of the *Endameba* were found in the pus.

The temperature dropped promptly, reaching normal within two days (Chart I) and never reached its original height; the pain was lessened and the patient's general condition

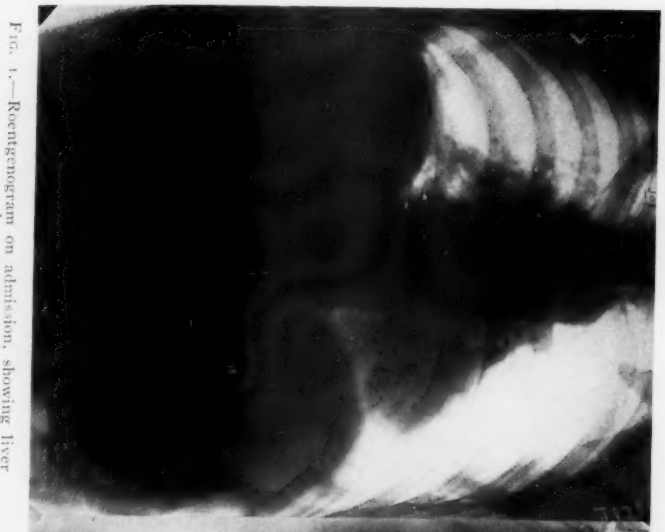


FIG. 1.—Roentgenogram on admission, showing liver enlargement.

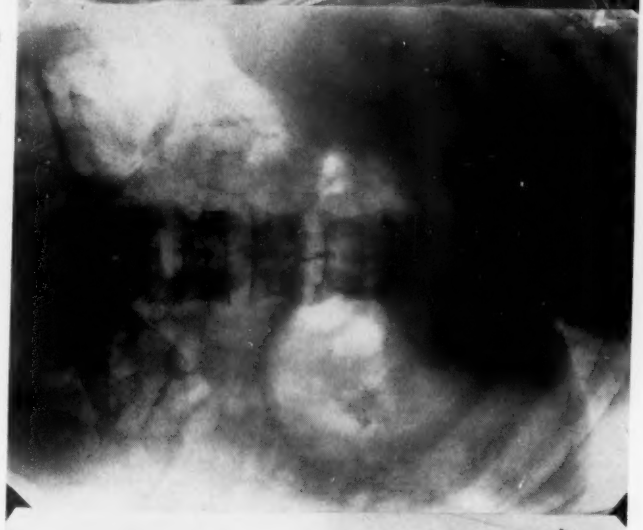


FIG. 2.—Roentgenogram after first aspiration. Less density of liver shadow.

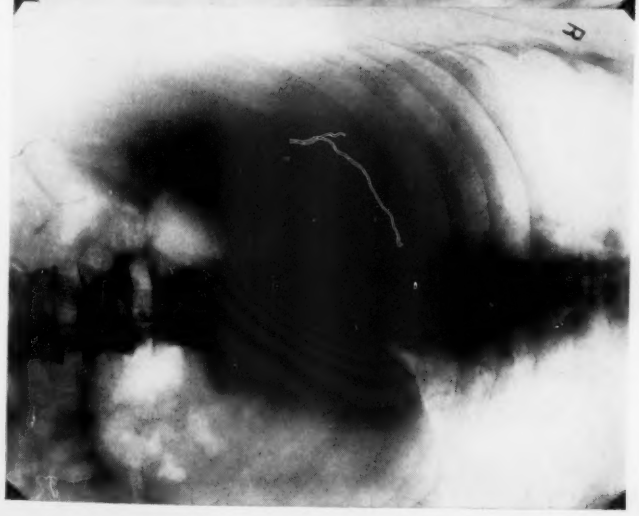


FIG. 3.—Roentgenogram after sixth aspiration.

improved. Five days later the liver increased in size, especially upward and to the left, and a pericardial friction rub was noticed. A second aspiration of the liver in the same area as before was made two days later, but no pus was located. After another five days the liver was again aspirated at the original site and ten ounces of pus removed, of a light brownish color, and not nearly so thick as that previously evacuated, was aspirated. The pericardial friction sound continued and after five more days the patient became slightly cyanotic. At a fourth aspiration of the liver ten ounces of thin chocolate colored fluid were obtained, after which the patient was much more comfortable. The liver was markedly reduced in size.

Nine days later, on October 6, just a month after admission, pain in the right chest appeared and the temperature rose to 101° F. The roentgenologic report was: "Liver shadow not so dense but the right chest is cloudy, suggestive of pleurisy with effusion." Paracentesis of the chest yielded four ounces of yellowish gelatinous fluid. Immediate relief of pain and a drop in temperature followed (Chart II). Three other aspirations of the liver were performed (Fig. 3), making seven altogether, one yielding eight ounces, one ten ounces, and the last none. During the period of the liver aspirations there appeared to be one large abscess, with possibly three or four smaller ones apparently coalescing with the main cavity as the treatment continued. Each time the needle was inserted in the eighth interspace, changing its direction somewhat after entrance, and two grains of emetine were introduced into the liver after each aspiration. One additional puncture of the right chest obtained no fluid. The patient was discharged October 28, 1934. He has remained well, and has had no return of symptoms to the present time.

Critique.—Two questionable points suggest themselves in the conduct of this case. First, it might be asked why we did not wait longer at the beginning to secure the systemic action of emetine before evacuating the abscess. It is agreed that the routine administration of emetine, one grain daily for ten days, should precede any operative procedure, provided the patient is seen early and there is time to wait for possible improvement. Undoubtedly the use of emetine (or some other drug of similar action) may prevent or abort the formation of an abscess in the liver at the very beginning. In the present case, however, the urgency was so great, and the abscess so large, that immediate evacuation was considered advisable, permitting a continuation of the emetine generally and locally.

The second question comes up in connection with the right pleural effusion. Was it present originally, as has occurred in other cases, and was it overshadowed by the liver enlargement? Or was it due to interference with the pleura in the various aspirations of the abscess? We think the pleurisy was a necessary complication in this delayed case, and we were not willing to aspirate the pleural cavity until the liver had receded sufficiently for differentiation. This could not have been brought about rapidly without at least partial evacuation of the liver abscess.

Abstracted Reports of Fellows of the Southern Surgical Association.—The subject of abscess of the liver, notably the amebic type, manifestly is of special interest to the Fellows of the Southern Surgical Association. Its membership embraces largely a region in which the affection is comparatively common and several of our Fellows have presented reports of their experience. This is the first case of the kind seen in our service, although one of us (H.A.R.) has operated upon three liver abscesses of metastatic origin, not

pleural effusion, and fifth and sixth aspirations of abscess.

Volume 103
Number 5

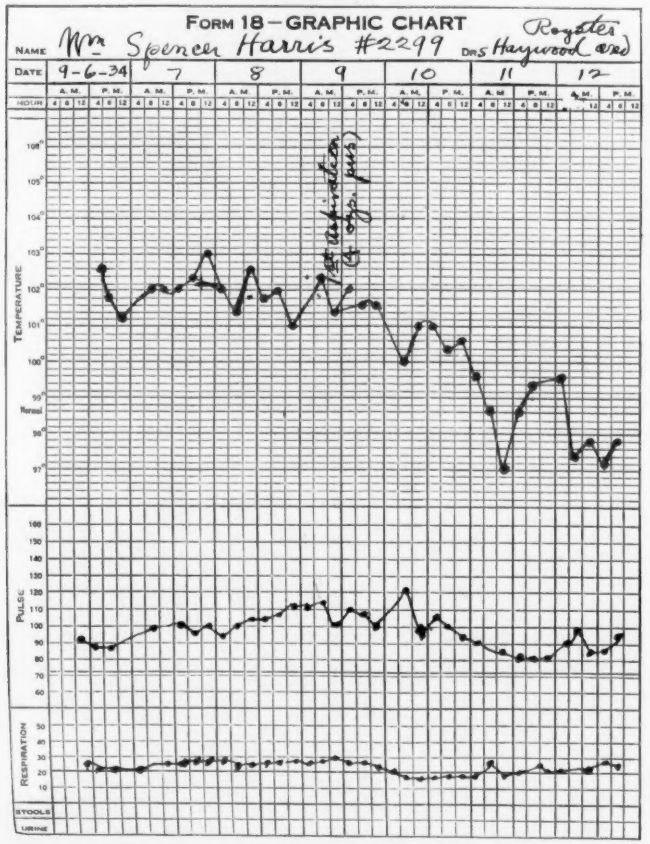


CHART I.—Temperature on admission and after first aspiration.

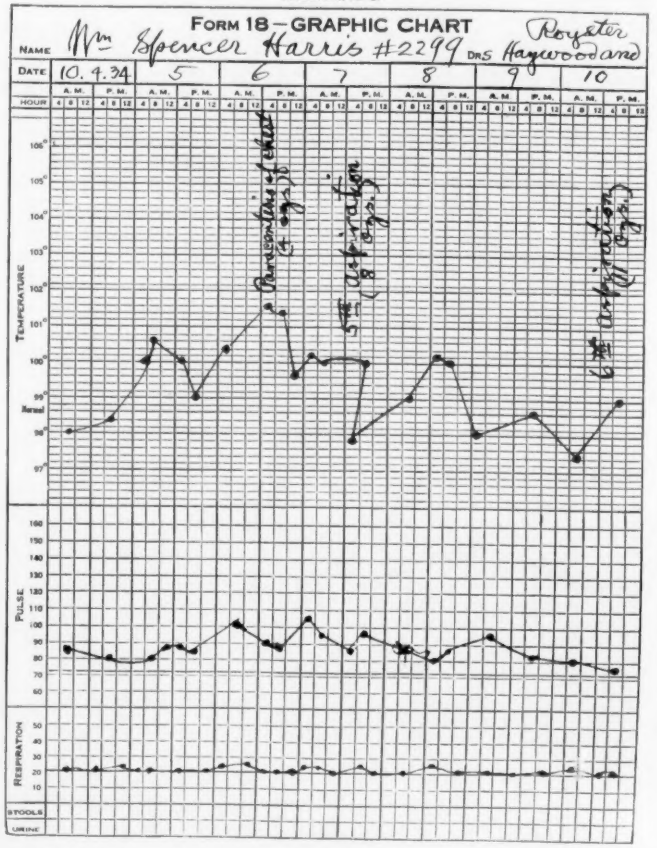


CHART II.—Temperature one month after admission and at time of tapping pleural effusion, and fifth and sixth aspirations of abscess.

caused by the *Endameba*. Parham, in 1922, reported an instance of abscess of the liver in a man aged 42, giving a history of frequent stools with blood and mucus for 21 months, who had been transferred from the medical division, where he had been under observation for eight days. "Amebae were suspected as the cause of the dysentery, but none were found," and "an exploring syringe was used in the eighth intercostal space, obtaining a thick, creamy pus, which showed in smear and on culture, the colon bacillus." The liver was enlarged more upward than downward, there was a high leukocytic count, and a temperature of 102° F. had been present, "though the febrile movement had not been marked." The diagnosis as made was "abscess of the liver, bacterial in origin, probably a sequence of the dystentery." The prognosis was considered unfavorable, as dealing "with a septic abscess rather than with the old classic, single, tropical abscess of Murchison, which we now know to be amebic." A transpleural operation was done, resecting the ninth rib posteriorly, opening the pleura, suturing the diaphragm to the muscular tissue and the liver to the diaphragm, evacuating the liver abscess and packing with iodoform gauze. "The pus at operation showed no organism and remained sterile on culture. It is probable, therefore, that the abscess was, after all, amebic and not a septic multiple abscess. We, therefore, gave him a few doses of emetine." The patient recovered but was still in the hospital at the end of the eighty-eighth day following his admission.

Boland (1931) had a series of 19 cases, 14 of the amebic type, with five deaths all presumably treated by open operation.

In 1933, Gessner contributed a report on 96 cases of true amebic abscess of the liver seen in New Orleans from 1918 to 1932 with a death rate of 39.58 per cent, with no differentiation of methods of management. But he quotes from the paper of Rogers a contrast of the open operation with 56.7 per cent mortality and the aspiration method with 14.4 per cent of deaths. Further, Rogers pointed out that his pupil Chathrji had treated 186 liver abscesses by aspiration with a mortality of 1.6 per cent.

Matas (1933) noted the gradually decreasing mortality of amebic liver abscess in later years and attributes it to earlier diagnosis, better types of patients and to the use of emetine along with repeated aspiration.

Hogan, in 1934, presented before the Southern Surgical Association a thorough review of the pathologic ravages of the *Endameba histolytica*. In reference to amebic abscess of the liver, he concludes, "If the emetine treatment hypodermically and aspiration and irrigation when indicated . . . secure the remarkable results . . . that have been reported, it is a remarkably good method of treatment." In the discussion of Hogan's paper, Noland expressed himself as distinctly opposed to the aspiration treatment of liver abscesses of amebic origin. He considers them frequently multiple, that aspiration of the dome of the liver is liable to infect the pleura, that often the puncture of the liver causes a leak into the abdominal cavity; and he strongly advocates abdominal exploration through a high incision in cases of suspected liver abscess, followed by rib resection, if needed, and open operation with drainage.

At that time Alton Ochsner called attention to the drop in the mortality rate from 47.2 per cent under open drainage methods to 6.9 per cent under conservative treatment. Later (1935) Ochsner and DeBakey published an exhaustive compilation of the literature of liver abscess, including a study of 73 cases of their own. Their "lowest mortality rate (4.1 per cent) was obtained in 24 cases treated conservatively by aspiration and the use of amebicides," and they concluded that "open operation should be employed only in those cases in which there is infection with pyogenic microorganisms."

Other Recent Reports.—In June, 1934, Brown, of the Mayo Clinic, reported 22 cases of damage to the liver from amebiasis, in 16 of which there was abscess formation, and in six others evidence of hepatic involvement—chills, fever, leukocytosis and tenderness in the region of the liver. One of the abscess cases, virtually moribund, died soon after admission, following open drainage. Autopsy revealed that the abscess had ruptured into the peritoneal cavity. "In the remaining 15 cases of known abscess, aspiration or open drainage had been done, accompanied by anti-amebic treatment. So far as we know all these patients have been cured."

Pauline Williams, in reporting an autopsy of a fatal case of amebic hepatic abscess, without previous dysentery or diarrhea, quotes Harrington as observing "a case of known amebic dysentery in which liver abscess developed even though the patient was given extensive treatment with emetine."

About two months ago (October, 1935) Holmes, of Chicago, published a paper on the medical treatment of amebic infections of the liver, reporting two cases of liver abscess, both treated with emetine alone, without either aspiration or open operation. Both patients recovered. In the first case a pleural effusion was suspected, but thoracentesis gave negative results. Under emetine therapy the patient recovered rapidly and was discharged at the end of 12 days. The second patient received the same treatment, but seems to have had a much slower recovery. Holmes voices strong objections to any surgical interference in amebic abscess of the liver, saying that both aspiration and open operation "should be reserved for cases complicated by secondary bacterial infections and for other complications, such as rupture of the abscess into the pleural or peritoneal cavities." He cites the following disadvantages of the aspiration method: that the abscess cannot be evacuated completely and the needle may cause dissemination throughout the liver, that no account is taken of multiple abscesses, which he thinks are not uncommon, that the parasite is not disturbed even by complete evacuation, since the amebae exist "at varying depths in solid liver tissue" and finally that "surgical treatment of an uncomplicated amebic abscess is illogical."

Comment.—Referring first to the conclusions expressed by Holmes, some of which are sound, we believe it proper to inquire how an abscess of the liver can be diagnosed with certainty unless the pus be demonstrated either by needle or knife. It must be remembered that acute hepatitis is the early manifestation of the liver invasion and that its symptoms are similar to, and often just as pronounced as, those which are observed when abscess super-

venes. As stated by Holmes, "the symptoms of the stage of abscess do not differ materially from those of the stage of hepatitis." It is granted that emetine should be used from the beginning in any suspected case, and that in many instances, abscess may be prevented or even aborted when it is seen in an early stage and is of insignificant size. But it would be unfair, we think, to assume the presence of a large abscess without actually seeing the pus, just as it would be improper to omit the routine use of emetine along with evacuation of the pus. Surely for diagnostic purposes, if not for therapeutic reasons, aspiration has a place, chiefly in the neglected cases, and especially in those of an emergency nature, requiring immediate relief from pressure symptoms in patients too ill to undergo an open operation and too far advanced to wait for the effects of an amebicide. Here may be reemphasized the injection of two grains of emetine through the aspirating needle after each withdrawal of pus, thus attacking the amebae in the depths of the liver tissues.

The criticism of Noland concerning infection of the pleura in aspiration of the dome of the liver has merit. It is not only possible, but quite probable; and yet this occurrence, in our opinion, is more than offset by the disasters, dangers and disabilities ensuing from the more formidable operation of incision and open drainage. The important answer to this objection is found in the overwhelming reduction of the mortality rate since the employment of the aspiration method. With care, contamination of the pleura apparently does not occur either in the introduction and withdrawal of the aspirating needle, or in the conduct of the open operation, the only difference being in the more serious character of the latter, with its exposure to outside infection and a longer period of confinement. Undoubtedly in many cases pleural effusion is present before any treatment is undertaken, as it has been shown that amebae may invade and penetrate the diaphragm from an abscess in the dome of the liver. It would appear that the effects of involvement of the pleura, which occasionally happens during aspiration of the liver abscess, have been exaggerated.

Summary.—In summarizing our own conclusions on the subject, we can do no better than to quote from Carson's Operative Surgery (1924) a paragraph written by G. Grey Turner: "The whole subject of the treatment of tropical abscess of the liver has been placed on a new footing since the value of emetine has become appreciated in the treatment of amebiasis. Our previous knowledge of liver abscesses was largely derived from cases much further advanced than are likely to be met with today, and the treatment was based on the assumption that the pus of such abscesses was pyogenetic in origin, whereas we now know that in the large majority of cases it is sterile. These abscesses are commonest in the right lobe, but in about 40 per cent of cases they are multiple. There is abundant evidence to show that such abscesses can spontaneously recover, and the aim of the treatment at the present time is to imitate the processes by which nature brings this about. One essential is to saturate the circulating lymph with emetine, which is inimical to the

ameba. In some cases this is all that is required, and the pus becomes innocuous, and either disappears or becomes inspissated. When further aid is necessary, the tension in the abscess must be relieved so as to encourage an outpouring of emetine saturated lymph from its walls and to allow of their collapse or retraction. This is done by the withdrawal of pus. The process may be repeated, but it is not essential that all the pus be removed. The routine treatment ought, therefore, to consist of (a) the intramuscular injection of emetine hydrochloride, 1 gr. daily for 10 days; (b) removal of the pus either with a large exploring syringe or an aspirator. The evacuation of pus may be carried out when the case is first seen, if there are urgent symptoms due to the size or situation of the abscess, but it is better deferred until a course of emetine has been administered. If there is evidence of secondary infection, or the case is not doing well, the operation of open incision may be subsequently required; but the preliminary treatment by emetine is necessary in all cases, and aspiration may further help to render open operation more successful."

REFERENCES

[1] Parham, F. W.: Surg. Clinics N. Amer., vol. 11, No. 5, p. 1295, 1922.
[2] Boland, F. K.: Annals of Surgery, vol. 94, p. 766, 1931.
[3] Gessner, H. B.: Am. Jour. Surg., vol. 20, p. 683, 1933.
[4] Rogers, L.: Philippine Jour. Sci., S. B., vol. 5, p. 219, 1910.
[5] Hogan, E. P.: Trans. South. Surg. Assn., vol. 47, p. 306.
[6] Ochsner, A., and DeBakey, M.: Am. Jour. Surg., vol. 19, p. 173, August, 1935.
[7] Brown, P. W.: J.A.M.A., vol. 105, No. 17, p. 1319, October 20, 1935.
[8] Williams, Pauline: South. Med. Jour., vol. 28, No. 10, p. 902, October, 1935.
[9] Holmes, W. H.: Surg., Gynec., and Obst., vol. 61, No. 4, p. 521, October, 1935.

Discussion.—Dr. Alton Ochsner (New Orleans, La.).—Doctor Royster referred to the group of cases which Doctor DeBakey and I reviewed. The relative proportion of amebic and non-amebic abscesses in the 102 cases considered was 71.5 per cent amebic and 28.4 per cent non-amebic. Amebae were found in 17.4 per cent of amebic abscesses and in 36.1 per cent of the stools of these same cases with amebic abscess. As regards the clinical manifestations, fever was present in 69.8 per cent and enlargement of the liver in 65.7 per cent.

The diagnosis of abscess of the liver is greatly facilitated by roentgenograms. As shown by Granger, a localized bulging into the lung field is usually indicative of hepatic suppuration, although it may rarely be the result of a gumma of the liver. As many cases of amebic abscess are seen after perforation into the subphrenic space has occurred, the roentgenogram is of inestimable value. In such an instance, in the anteroposterior view there is elevation of the medial portion of the right diaphragm with obliteration of the cardiophrenic angle, whereas that taken from the side shows an elevation of the anterior portion of the diaphragm and obliteration of the anterior costophrenic angle. On the other hand, in pyogenic subphrenic infections, which are usually secondary to appendiceal infections, in the anteroposterior roentgenogram there is elevation of the lateral half of the diaphragmatic leaf with obliteration of the costophrenic angle and in the lateral view there is elevation of the posterior portion of the diaphragm with obliteration of the posterior costophrenic angle.

In 16 cases treated by transpleural drainage there was a 25 per cent mortality. In 14 cases treated by a right rectus incision there was a 21.4 per cent mortality. In seven cases treated by incision and drainage over the most prominent presenting portion there was a 14.2 per cent mortality. In nine cases treated by incision through the retroperitoneal approach there was 11.1 per cent mortality, whereas in 24 cases treated by aspiration and emetine there was a 4.1 per cent mortality. The combined mortality in the entire group of 70 cases was 14.2 per cent.

We feel that emetine should be used with extreme caution. It is a dangerous drug, and the total amount given should never exceed 10 grains. Other amebacides should not be used, because they are liver poisons and exert little effect on the amebae in the liver. As most amebic abscesses are sterile (89 per cent of our cases), open drainage is undesirable because of the danger

FIG. 4.—The sectioned surface of the liver, through five of the amebic abscesses. One near the diaphragmatic surface was drained (Dr. Louis Frank).

of secondary infection which markedly increases the mortality rate. Conservative treatment consisting of aspiration and emetine administration is the treatment of choice. We feel that these abscesses are analogous to tuberculous abscesses, and the patient who dies does so as the result of secondary infection.

DR. LOUIS FRANK (Louisville, Ky.).—Not living on the Southern coast but inland we have seen but few cases of amebic abscesses until the recent endemic which had its origin in Chicago. Since that time we have had an opportunity of seeing quite a few. Early in the epidemic there was some confusion in diagnosis and several cases were operated upon as appendicitis and one for supposed malignancy of the cecum, all which ended disastrously. Our own experience with abscess of the liver of amebic origin dates back 30 years. The patient had never been out of the State, and had a single

abscess of considerable size in the right lobe of the liver. He recovered following an open operation.

Among the cases arising from infection in Chicago there were two with liver abscess. One, unfortunately, was multiple from the beginning and presented symptoms of such acute character that we thought immediate operation was demanded. Two abscesses were identified and drained. The patient died 48 hours subsequently. He had been under active treatment with emetine for five days preceding his operation. At autopsy seven abscesses were found in the liver (Fig. 4). It is quite possible that this patient might have done better if the emetine had been continued and had he not been operated upon, but the symptoms were so acute that we believed intervention was demanded. The cecum was studded extensively with amebic ulcers and was much thickened. The ileocecal valve was so markedly involved that it was held patent, as demonstrated by the barium enema.

The other patient we saw with liver abscess was a boy, who possibly had an abscess present at the time of his admission to the hospital. The case was diagnosed as acute appendicitis and the appendix was removed. The temperature continued and he developed other more definite symptoms of liver abscess. His first abscess was in the left lobe of the liver. This was opened and drained. He improved but later developed an abscess in the right lobe which in turn was also drained. This was followed by a massive lung collapse, which under a tent with oxygen and CO_2 gradually cleared up, but was shortly followed by a lung abscess necessitating the resection of a rib. He was in the hospital four months, but finally made a good recovery.

A diagnostic point which we would like to call to your attention, and which we think can usually be elicited, is true and definite finger point tenderness. This we believe is a very important diagnostic finding, and with careful study of the case, and the other symptoms which present, should enable one to make a diagnosis.

We believe, as Doctor Ochsner has just mentioned, that these cases of abscess of the liver occur chiefly in those individuals having a preponderance of the amebic infection located in the right colon and in the cecum. We also believe the cecum will show in such cases, with or without abscess of the liver, some very typical findings. Usually there is a shortening of the cecum, apparently the result of the infiltration and an ileocecal incompetency. This condition may easily be mistaken for a new growth or even for a tuberculous process.

Dr. Hubert A. Royster (closing).—I believe with Doctor Ochsner that as a rule one aspiration in a case is sufficient if seen early. The repeated aspirations in our case were carried out because of the continued pain and swelling in spite of the drop in temperature. The needle was inserted in different places. We had the feeling that there were abscesses in various places, all in the right lobe. The fact that we had a pericardial friction sound might lead one to think that there might have been abscesses in the left lobe also, but continued emetine therapy, we felt, would take care of them. We know that 40 per cent of these abscesses are multiple, but there is no way of finding that out in the beginning, and it seems to us the repeated aspirations for relief are safe. After each aspiration we also injected two grains of emetine into the abscess cavity through the needle. I believe this is important, for if there is any value in emetine we obtain it both through the circulation and in the abscess cavity. Of course, the regenerative powers of the liver are well known. I think if I had a similar case, in which we felt that multiple abscesses were actually present, we would use the same treatment.

ABDOMINAL GAS BACILLUS CATASTROPHES

ROBERT L. RHODES, M.D.
AUGUSTA, GA.

GAS bacillus infection in several of its aspects has been presented before this association during the past several years, *i.e.*, in compound fractures and postoperative drainage wounds, which have been associated with injuries or perforative or suppurative abdominal lesions. It is my purpose to offer for discussion, founded upon two cases, gas bacillus infection of the intestinal tract and peritoneum in which there was no evidence of perforation or suppuration. Whether or not they should be classed as gas gangrene of the gastro-intestinal canal (small bowel) or gaseous peritonitis, remains for your decision. This title was selected as a statement of facts, rather than employing either of the above captions, both of which are, at least, controversial. In all the literature, there has been no attempt to separate those cases not associated with perforative or suppurative processes within the abdomen from those associated with such processes, therefore it has been necessary to review the subject in its entirety.

CASE REPORTS

Case 1.—A colored male, aged 35, night watchman, had had a right inguinal hernia for several years. At 4 A.M. while going down some steps his hernia slipped down, which was nothing unusual, therefore he continued his duties until 7 A.M. when he checked out. Failing in his attempts at reduction he went to a doctor, who was also unsuccessful after a few minutes of taxis and he was sent to the hospital. He arrived at 10 A.M., having vomited several times on the trip, a distance of 65 miles, no vomiting before. Physical examination showed a very muscular man, with a large mass of strangulated hernia, right indirect inguinal, its size suggesting two or three feet of incarcerated bowel. Temperature, 97.8° F.; pulse, 64; white blood count, 10,000; polymorphonuclears, 80 per cent; hemoglobin, 80 per cent; urine negative except for diacetic acid one plus. Preparation for immediate operation was made, sodium amytal Gr. 6, morphine Gr. 1/4 and atropine Gr. 1/150 administered.

Operation.—Under ether anesthesia an incision was made over the inguinal canal. As the peritoneum was opened above the neck of the sac there was a gush of chocolate or coffee colored fluid with an exceedingly foul odor, a pint or more escaping. This odor impressed the entire operating team with its foulness but attracted no more attention at this time. The neck of the sac was incised and about three feet of strangulated bowel withdrawn, several ounces more of the same type fluid escaping. No gas was recognized in the fluid. The strangulated bowel was quite dark; it was carefully examined for any evidence of perforation, but none was found. The viability of the bowel was questionable, but after ten minutes of applications of hot saline packs its color improved markedly and the mesenteric vessels showed no evidence of thrombosis, therefore, it was felt safe to replace it and close the hernia. Four hundred cc. of 10 per cent glucose were given intravenously on the operating table, because of the history of vomiting and the presence of diacetic acid in his urine. He stood the operation well, left the table in excellent condition and reacted normally from the anesthetic. During the afternoon glucose was repeated, as a safety precaution rather than because of any definite indication for it.

At 4 P.M. he expressed himself as feeling so well he was ready to go home. The temperature was 100° F., pulse 130, for which no cause was found. At 7 P.M. the nurse gave him nourishment and was leaving the room when she heard a groan and on looking back saw him throw up his arms, groan again, turn partially over and die at 7.10 P.M. Such a death would have ordinarily been thought to have been due to a pulmonary embolus.

Autopsy by Dr. Edgar R. Pund, 18 hours postmortem: The abdomen was considerably distended, the peritoneal cavity contained much foul smelling gas and some 1,000 cc. of bloody fluid, the serous surfaces were dull and greenish and there was an occasional shred of fibrin. There was gas in the liver, retroperitoneal fat and blood vessels. The small intestine was considerably distended with gas, necrobiotic changes throughout, much more marked in the loops which had been strangulated in the hernia. All tissues and organs were dull and cloudy, sometimes discolored greenish and much gas bubbled from the vessels. The anatomic diagnosis was necrobiosis of intestines, peritonitis, gas bacillus infection.

Case 2.—White male, aged 55. Past history, irrelevant other than that he had always been a heavy meat eater. During the past three years he had had frequent attacks suggesting angiospasm of the right leg, with cramps in the calf which after a short rest would relax. He had had several attacks during the few weeks preceding his present illness, one involving the right hand from which he had not fully recovered and had had no palpable radial pulse since. His present illness was of acute onset and followed a day during which he had felt unusually well. It began at 9.30 P.M. with a sudden pain in the epigastrium, for which he took soda but without relief, the pains soon becoming generalized over the entire abdomen. There had been no dietary indiscretion during the day and his bowels had moved freely three times. The pains became steadily worse during the night; he employed three enemata but only the water returned and without relief of the pain. He was unable to lie down but had to sit in a chair and lean forward, could not lean back. He was in a drenching perspiration all night. About 2 A.M. he began to complain of abdominal distention, took several glasses of hot salt water at half hour intervals, which was regurgitated. He eructated gas in large quantities constantly throughout the night, especially after his abdomen began to swell, but it afforded no relief. Temperature, 96° F. at 6 A.M. Several doses of codeine were taken without any relief; these were retained, as he vomited only after taking the salt water. A cup of hot coffee at 7.30 A.M. was promptly vomited. His distention increased steadily after 2 A.M. and caused more and more difficulty in breathing. At 8 A.M. he went out on the porch to get in the fresh air because of his difficulty in breathing and feeling of suffocation. During the night he had been waxy pale but after daylight a purplish, livid color became progressively more evident. I saw him at 8.30 A.M., sitting in a chair on the porch, cyanotic, gasping for breath, eructating gas in enormous quantities constantly, his abdomen was fearfully distended, tympanitic everywhere, liver dulness obliterated, generalized tenderness and rigidity, a trifle more marked over the right lower quadrant. Temperature, 100° F., pulse, 130. On suggesting an operation, he said, "Well, let's hurry because if you don't soon put a hole in there I'll bust open." His condition became rapidly worse and his color more livid. Temperature, 99.2° F.; pulse, 114; white blood count, 31,800; polymorphonuclears, 89 per cent; urine showed four plus sugar. Blood sugar, 200; N.P.N., 22.4; Wassermann and Kahn tests were later reported as negative. His distention became even more marked, color more cyanotic, and his heart quite embarrassed by the distention. Acute hemorrhagic pancreatitis, in all its violence, seemed the most probable diagnosis.

Operation.—Upon opening his abdomen there was an escape of several ounces of coffee colored fluid with a very foul odor but no gas was recognized. There was no evidence of fat necrosis to suggest pancreatitis. The entire small intestine was distended almost to the point of rupture, the musculature presented striae similar in appearance

to striae albicantes of the skin, several loops were hemorrhagic, and gave very much the appearance of a loop freed from a strangulated hernia. The colon was not distended. It was apparent we were dealing with a gas bacillus infection. Search did not show any evidence of perforation anywhere or acute inflammatory process in appendix or in any other organ. Blood cultures were made which later showed no growth.

Autopsy.—Two hours postmortem: On opening the abdomen the characteristic gas bacillus odor was remarked. Coffee colored fluid, as previously described, and in addition some gas were evacuated. Cultures were made which showed the *Clostridium aerogenes-capsulatus* (Welch). Smears also showed this *Clostridium* and only a very occasional cell. A closer, more detailed search failed to show any point of perforation in the gastro-intestinal tract or evidence of any inflammatory process in appendix, pancreas, liver, gallbladder or elsewhere than in the small intestine where these mottled, congested, hemorrhagic edematous loops were found, the intestine when opened showed no evidence of ulceration anywhere, bubbles of gas were seen in the walls of these loops.

Probably the most exhaustive review of the subject in all its phases is that by W. Lohr,[1] in 1929, which I shall abstract freely. The relationship in perforative lesions of the digestive tract is well established, but this is not true as regards the action of anaerobes as a source of peritoneal infection or the course of anaerobic peritonitis. "Gas Peritonitis" refers to a very peculiar, typical, and apparently rare, disease picture usually following laparotomy. Lohr's description of the course is quite similar to that of Case 2, except he states the intestines are compressed and collapsed by the gas in the abdomen. Gross inflammatory changes in the peritoneum and the intestines are lacking. He refers to one case arising spontaneously in which at operation, the peritoneal cavity was found filled with gas, the intestines normal, the appendix was removed, which histologically showed acute appendicitis and no other pathology found. The patient recovered. (Probably Michejda's[2] in which the gas was odorless and had nothing in common with anaerobic gas bacilli.) In all cases reviewed by him bacteriologic examination had not been made. He recognizes that bacteria other than anaerobes can cause gas. In reliable pure culture anaerobic infection of the peritoneum of man and animals the peritonitis began with an exudate poor in cellular constituents and pronounced intestinal paresis and not with excessive accumulation of gas. A gas edema or a gas phlegmon of the stomach and intestine, corresponding to the progredient process of a muscular gas edema, has never occurred. Much larger doses of anaerobes are required to produce a peritoneal infection than a muscular infection and the peritonitis was always without gas formation. His explanation is that gas occurs only when the anaerobic bacilli can produce necrosis in the tissue infected and then produce gaseous decomposition. The fine distribution of the toxin in the peritoneum and the excellent blood supply allows the formation of a toxin to occur which can lead to a dangerous toxic effect, which can be expressed only in capillary paralysis with large exudation of a pale to a very hemorrhagic exudate with but few if any leukocytes. Thus one must assume that gas edema bacilli are not causes of inflammation but only form toxin. The nutri-

tion of the intestines in man and the experimental animal is so secure that there is neither necrosis nor gaseous disintegration of these organs during life.

Again in 1933 Lohr,[3] criticising the report of a case of "Gas Gangrene of the Gall Bladder" (Koch[4]), states that in gas gangrene of the liver (gall-bladder), anaerobic peritonitis and gas gangrene of the intestines do not exist, as he could show by numerous references to the literature as well as in his own animal experiments, which has also been proven beyond doubt by Lampa and Galavotti's bacteriologic experimental work.

On the other hand, W. Plath,[5] in 1934, collected ten cases of gaseous peritonitis in the literature and classified them in three groups. (1) Following operative procedures within or outside the peritoneal cavity, apparently there are no inflammatory signs in the peritoneum. (2) Spontaneous, when apparently an inflammatory factor is present. (3) From the gastro-intestinal tract through a perforation or intact walls. He reports a case in the third group, but since a mass was palpable in the upper abdomen, after a fistula was established, the gas was probably secondary to perforation. An autopsy was not permitted.

Lamprecht,[6] in 1928, states that primary gas gangrene of the gastro-intestinal canal is very rare but does occur, and refers to five cases described in the literature. (C. Fraenkel, Hitschman and Lindenthal, K. Jeppson, Kirshmeyer, and Bruett.) Gas gangrene infections after operations upon the intestines are also rare, under either septic or aseptic conditions. Brentano observed two cases and Flechtenmacher one case after herniotomies. He reports a case of gas gangrene of the abdominal wall and death 40 hours after operation for old existent appendiceal abscess with marked obstruction of the terminal ileum in which enterostomy at this point was performed at the same time. Autopsy revealed advanced gas gangrene of the entire abdominal wall and of the piece of ileum used for the fistula, the latter only as far as the muscularis was concerned. There was a large amount of hemorrhagic serum in the abdominal cavity, from which the *Clostridium welchii* was cultured as well as from the tissues.

Nason and Starr,[7] in 1934, presented an excellent general discussion of the subject of "Gas Bacillus Infection Complicating Laparotomy" in which they state that the infection may begin in the muscle of the abdominal wall, the peritoneal cavity or the liver: that the pathologic changes are the result of the toxemia alone or plus the presence of organisms and gas.

Maguire,[8] in 1934, says gas gangrene may occur subsequent to appendectomy, herniotomy and other operations performed on organs in close proximity to the intestinal tract, in which instances the causative organism gains entrance to the wound from the alimentary canal.

Finesilver,[9] in 1934, in his discussion refers also to the studies of Weinberg and Seguin that the *Bacillus aerogenes-capsulatus* was in association with other bacteria, and states that the combined efforts of symbiosis of several species of bacteria are required to produce gas gangrene, the saccchar-

olytic group starting and the proteolytic group completing the destruction. The *C. welchii* elaborates a hemolytic toxin which produces further local destruction of tissues, and by absorption, remote constitutional symptoms.

C. Knapper,[10] in 1929, presented a general survey of the subject, and comments that because gas edema bacilli and spores are found in the healthy gastro-intestinal canal, their development and toxin formation are especially to be feared in ileus. He refers to a fatal case of gas edema of the sigmoid under his personal observation but gives no details. Among the various ways of endogenic infection, he mentions by way of a typhoid or tuberculous ulcer of the intestine or through infection of the abdominal cavity itself with gas edema bacilli, after intestinal perforation.

All commentators agree that when the infection invades the blood stream, the peritoneum or the liver, the outcome is fatal. The consistent finding of extensive necrosis of the suprarenal glands may account for the rapid fall of the blood pressure, so characteristic of gas bacillus infection. A very high leukocytosis and an unusually rapid pulse, out of proportion to the temperature, or other findings, is the rule. That the *C. welchii* may reach the blood stream from any focus anywhere in the body is undoubted. That devitalized muscle is necessary for its growth and development in the tissues. That it may develop in the peritoneal cavity from perforation or operative soiling from the intestinal tract seems well proven. That toxemia from its development in the intestinal tract in cases of ileus may be quite profound even fatal.

Since there is muscle tissue in the wall of the intestine, is it not reasonable to assume that it may become traumatized and its blood supply markedly impaired by either strangulation as in a hernia or extreme overdistention from gas or other causes? Cannot such traumatized muscle itself become a site for the development of the *C. welchii*, from which site the organisms may permeate, rather than perforate, into the peritoneal cavity, and there produce gaseous peritonitis or may invade the blood stream for general dissemination? The toxins are so lethal, no large area of primary focus is required to produce a fatality.

Both cases, herein reported, would seem to qualify under these conditions as true cases of primary "gas edema" of the intestinal tract with gaseous peritonitis, since both showed gas bubbles in the musculature of the intestinal walls; one cultured the *C. welchii* from the peritoneal fluid and showed it in smears, the other (not cultured) showed the same characteristic chocolate or coffee colored foul peritoneal fluid; one had known trauma of the intestine with dynamic ileus (strangulated hernia) of seven hours duration, the other had an adynamic ileus of 12 to 15 hours duration, cause undetermined; one died apparently from a blood stream infection, possibly gas embolus, the other a typical death from gas bacillus toxemia.

Whereas, no gross perforations were apparent to the naked eye, it is perfectly possible that microscopic perforations may have been present in both cases. Such could have occurred by gas bubbles breaking through the

peritoneal coat from its point of origin in the muscularis and carried with it the organisms, since these have been shown to be present in the gas bubbles *in situ*.

Is it not possible that we are overlooking more of these cases by incomplete bacteriologic studies begun at the time of operation, by too few autopsies and failure to make bacteriologic studies in conjunction with the autopsy?

NOTE.—I wish to express my appreciation of the help of Dr. E. S. Sanderson, Professor of Bacteriology in the University of Georgia, School of Medicine, especially in looking up the literature and other suggestions.

REFERENCES

[1] Lohr, W.: Zentralbl. f. Chir., vol. 56, pp. 774–779, March 30, 1929.
[2] Michejda, K.: Zentralbl. f. Chir., vol. 54, p. 1871, 1927.
[3] Lohr, W.: Zentralbl. f. Chir., vol. 60, p. 2435, October 14, 1933.
[4] Koch, Friedrich EL.: Zentralbl. f. Chir., vol. 60, p. 2060, September 2, 1933.
[5] Plath, W.: Zentralbl. f. Chir., vol. 61, p. 1878, 1934.
[6] Lamprecht, H.: Arch. f. klin. Chir., vol. 150, p. 328, April 12, 1928.
[7] Nason, L. H. and Starr, A.: Arch. Surg., vol. 29, p. 546, October, 1934.
[8] Maguire, C. E.: Jour. Mich. St. Med. Soc., vol. 33, p. 196, April, 1934.
[9] Finesilver, E. M.: Jour. Med. Soc. of N. J., vol. 31, p. 194, April, 1934.
[10] Knapper, C.: Nederlandsch Tijdschrift voor Geneeskunde, vol. 73, p. 4792, October 12, 1929.

DISCUSSION.—DR. WILLIS G. GATCH (Indianapolis, Ind.).—A number of years ago I was asked to remove a node from the groin of a nurse who had arthritis. I did that under the most rigid aseptic technic but the pathologist reported a growth of *C. welchii*. This struck me as a very peculiar finding in a node not the seat of any particular inflammatory disturbance. You will remember that a few years ago there was considerable discussion in the literature following the work of Andrews of Chicago on bile peritonitis. I think that studies in our laboratory will go far toward clarifying this condition. These have shown that in the liver of old dogs there is nearly always present an anaerobic organism which cannot be distinguished from the *C. welchii* by the smear, but which is distinctly different in that it is not fatal to laboratory animals when injected intravenously. It is occasionally found in the liver of elderly human beings. In the muscle of dogs is found a similar organism, also non-pathogenic, but somewhat different from that in the liver. This is also found occasionally in the muscles of human beings. I think this organism is responsible in some cases for supposed *C. welchii* infection with no gangrene and followed by recovery.

The gas bacillus is present in the intestine of human beings, and I am inclined to believe that the cases reported by Doctor Rhodes are due to the true organism.

I saw a case of peritonitis from gas bacillus in which the abdomen was collapsed. One of our gynecologists had done an hysterectomy. The patient had not done well and about the third day I was asked to see her. The abdomen was tremendously distended, and the liver dulness absent. I was asked to operate, which I did under local anesthesia. As soon as I opened the abdomen it collapsed and the intestines were found deflated. The surgeon had removed the appendix before he performed the hysterectomy. I think when this is done one should always perform the hysterectomy first and the appendectomy afterward.

Dr. H. J. Boldt (New York, N. Y.).—Some 40 years ago I performed an ovarian operation upon a woman; on the second day I found the patient's abdomen very much distended. There was no rise in temperature but the pulse rate was increased. It did not seem to me from her appearance, with no elevation of temperature, that she had a peritonitis, and I asked my friend Doctor Garrigues what he thought about it. He said it was probably a gas bacillus infection and advised reopening the abdomen. This advice was followed and a large amount of gas escaped. We put a tube in and the patient made an uninterrupted recovery. I have not previously reported the case because no examination of the bacillus was made.

Dr. Charles W. Flynn (Dallas, Tex.).—In view of a very recent case of gas bacillus infection of the abdominal cavity, which came under my observation, I was very much interested in the report of Doctor Rhodes' cases. Gas bacillus infection of the gastro-intestinal tract or following operations on the gastro-intestinal tract has been very rare in my experience.

Case Report.—On November 13, 1935, a white male, aged 35, entered Baylor Hospital, complaining of nausea, vomiting, loss of weight and weakness for the past two and one-half weeks. For ten weeks he had complained of his stomach. The laboratory findings showed a low gastric acidity, positive Wassermann and Kahn tests, blood in the stools, and in the gastric contents. Physical examination revealed a general adenopathy, the heart was slightly enlarged, the lungs were negative. Blood pressure, 132/72. The abdomen was moderately distended with gas and there was tenderness over the epigastrium. The prostate was moderately enlarged and tender. There were external hemorrhoids. There were no pathologic reflexes. His general condition at this time was considered fairly good, although he had lost 15 pounds in the last 20 days. Gastro-intestinal roentgenologic studies revealed a lesion of the lesser curvature of the stomach, diagnosed as a carcinoma. Blood examination showed 6,300 white cells, 68 per cent polymorphonuclears. Hemoglobin was 66.9 per cent.

Progress.—While the patient was in the hospital he continued to vomit his meals. Temperature, pulse and respiration normal. He was placed on belladonna, 20 minims before meals, a bland diet and frequent feedings. His condition did not improve. However, he was still considered a good operative risk. After careful preoperative care, he was subjected to a partial gastrectomy. Two-thirds of the stomach was resected. Immediate postoperative condition, fairly good.

About 24 hours after operation the patient's condition became critical, with a rapid, thready pulse, shallow and rapid respiration, and slight cyanosis. He was given glucose intravenously, morphine and caffeine sodium benzoate hypodermatically, and was placed in an oxygen tent.

Physical examination showed numerous coarse râles throughout the lungs and his abdomen became markedly distended. The pulse increased, his temperature reaching 102° F., and he expired 36 hours after the operation. Nine hours after death a complete autopsy was performed by Dr. W. W. Brandes, with findings as follows: (1) Primary cause of death, gas infection (*C. welchii*). (2) Secondary cause of death, gastric resection for carcinoma. A brief synopsis of the autopsy report was: 1.—Recent gastric resection. 2.—Generalized fibrinopurulent infection—gas formation. 3.—Generalized invasion of body by gas forming organisms. 4.—Generalized hemolysis of blood. 5.—Generalized softening of tissues, especially the viscera.

Dr. Urban Maes (New Orleans, La.).—I should like to make one or two observations that bear on Doctor Rhodes' contribution. You will remember that when Dragstedt of Chicago planted free fetal liver in the abdominal cavity of animals there were no untoward results, though the typical picture of peritonitis followed when any but fetal liver was implanted, a

result that is highly suggestive. At present Dr. Frederick F. Boyce is conducting some experiments on autolytic peritonitis. All of the dogs in which he implanted liver, that had not been autoclaved, died promptly, with the typical picture of peritonitis seen under such circumstances. Two of the dogs, however, in whom we implanted liver, that had been sterilized (in one case unground and in another ground), both lived and showed no ill effects. Clearly the organisms described as *C. welchii*, or gas bacilli, are a normal inhabitant of the liver, and, equally clearly, they can be destroyed by sterilization.

From the clinical side, my own experience is that tachycardia is one of the most helpful diagnostic signs in this condition, often appearing before other evidences of the infection are manifest. I am always inclined to view, with grave suspicion, any patient in whom the circumstances are propitious for this type of infection and who develops a rapid pulse that cannot be otherwise explained.

It is unfortunate, as others have pointed out, that the term "gas bacillus infection" has come into such general use. It conveys a totally wrong impression. Doctor Finney will bear me out, as will any other surgeon with war experience, that when wounded soldiers exhibited gas in their wounds, it was frequently too late to do anything for them. On the other hand, when we saw them before crepitation was apparent, the prognosis was not always so hopeless.

DR. ROBERT L. RHODES (closing).—I think Doctor Gatch overlooked the fact that in the second case the *C. welchii* was demonstrated in the culture. It was the first case that made us proceed carefully in the second.

The point I wished to bring out is that such catastrophes should be very carefully investigated. Doctor Lahey reported yesterday two cases of gas bacillus infection from dilating the sphincter of Oddi. These occurrences are becoming more frequent and if we are to get anywhere we must keep their etiology in mind.

INGUINAL HERNIA

Ross A. Woolsey, M.D.
St. Louis, Mo.

Webster defines rupture as a breaking apart or a state of being broken apart. The term rupture is objectionable since it implies an opinion as to the mode of formation. Hernia is a protrusion of a viscera from its cavity, and is the sequel of a congenital defect often made apparent by effort. Colcord[1] states that the fact that hernia is first complained of by the patient after an accident or a heavy lift does not prove that the accident caused the hernia. It discloses it to the patient, if he is honest, or if he is dishonest it affords him an opportunity to sell an old hernia to his employer. The profession has not properly impressed on the laity the knowledge developed by operation. It is now generally conceded by students of the subject that traumatic hernia can be caused only by direct violence producing a tear of the wall followed by a protrusion of the viscera at the point of violence.

The inguinal hernia is of two types, direct and indirect. It is quite apparent that the direct hernia has no connection with either the internal or the external ring, under which condition it is fair to assume that a weak posterior canal wall is responsible for direct inguinal hernia. In the etiology of indirect inguinal hernia, authors have considered the persistent processus vaginalis, the lax internal ring and the lax external ring. According to Halsted, the cord is the first cause of hernia, and the ultimate obstacle to its cure; that removal of a large part of the cord greatly facilitates its cure. Yet it has been conceded by many that the transversalis fascia is the sole important structure needed to retain the viscera within the abdomen. We believe that the external ring is in no way responsible for inguinal hernia. The large internal ring is the result of a weak posterior canal wall. The tissue above the internal ring takes no part in hernial formation and should not be sutured.

Brown[2] reports a case of a jockey whose horse fell on him. He became immediately ill with severe pain, vomiting and collapse. A few minutes later a typical small strangulated hernia was found and reduced by taxis. There was no previous history of hernia, yet subsequent operation demonstrated a small sac containing adherent omentum. Operative findings not infrequently demonstrate whether the hernia is recent or old.

Thirty-five years ago Russell[3] discussed the persistent processus vaginalis and concluded that an oblique inguinal hernia never occurs independently of the presence of a congenital sac. His point is not well taken, else recurrence would not occur after proper removal of the sac. Dissections of inguinal canals without hernia have developed unobliterated sacs in about 25 per cent of the dissections. This also would lead us to believe Russell's point was not a valid one.

Bassini, in 1889, gave to the profession an operation that can be classed as a radical cure. Removal of the sac, which up to this time had not been attempted, was the keynote of his success. He attached the internal oblique transversalis muscle and conjoined tendon to Poupart's ligament, transplanting the cord over the newly formed pad, then suturing the divided aponeurosis, thus recognizing the posterior wall defect. Ferguson first severely criticized Bassini's transplantation, dropping the cord against the posterior wall, then followed Bassini's technic. Later Pitzman[4] advised that the inguinal canal should be anatomically restored.

Andrews[5] modified Bassini's operation, in that he retracted the internal oblique and transversalis muscle, suturing the transversalis fascia to Poupart's ligament. We believe that this is inadequate, since the fascia is normally thin medially to the iliac vessels and on account of the direction of the fibers will not hold a suture properly.

A number of surgeons have used the fascial suture since first advocated by McArthur,[6] in 1902. It can be used only as a continuous suture in muscles sutured to fascia, which we believe inadequate.

We believe that the inclusion of muscles in hernia sutures is not warranted. The ease with which the aponeurosis is stripped from muscle should be proof enough that the muscle tissue will not permanently attach itself to fibrous tissue and since we became definitely convinced that a weak congenital posterior inguinal canal wall is responsible for both direct and indirect inguinal hernia, we developed a procedure in 1920 that has proved satisfactory.

Seelig,[7] in 1924, drew attention to the fact that muscle tissue does not unite to fascia, when these two structures are coapted by suture, in the sense that fascia unites to fascia. His experiments developed the observation that when muscle masses are united to white fascia actual union occurs only between the white fascia and connective tissue elements forming the septa between bundles of red muscle tissue. It has been shown that fascia will unite to fascia under considerable tension; however, under spinal anesthesia, the abdominal muscles are completely relaxed and all tension is relieved by jack-knifing the operating table and is maintained by keeping the bed jack-knifed, plus adequate opiates, for the first week following the operation.

Operative Technic.—The skin, superficial fat, and fascia are incised from above the abdominal ring to below the superficial ring, extending directly over the canal. By gauze dissection it is stripped from the external oblique aponeurosis. The superficial ring is cut with blunt scissors on its medial side and the aponeurosis slit to above the abdominal ring and separated from the deeper structures laterally, affording an excellent exposure of Poupart's ligament and medially the rectus sheath is exposed. The cord is next lifted from the floor of the canal close to the spine of the pubis. The entire content of the canal is lifted and dissected from its floor upward and outward to the abdominal ring. The hernial sac is next identified and opened. A finger is inserted in the sac as a guide when it is separated from the

FIG. 1.—Showing position of incision through the external ring, on its medial aspect, and its continuation through the aponeurosis above.

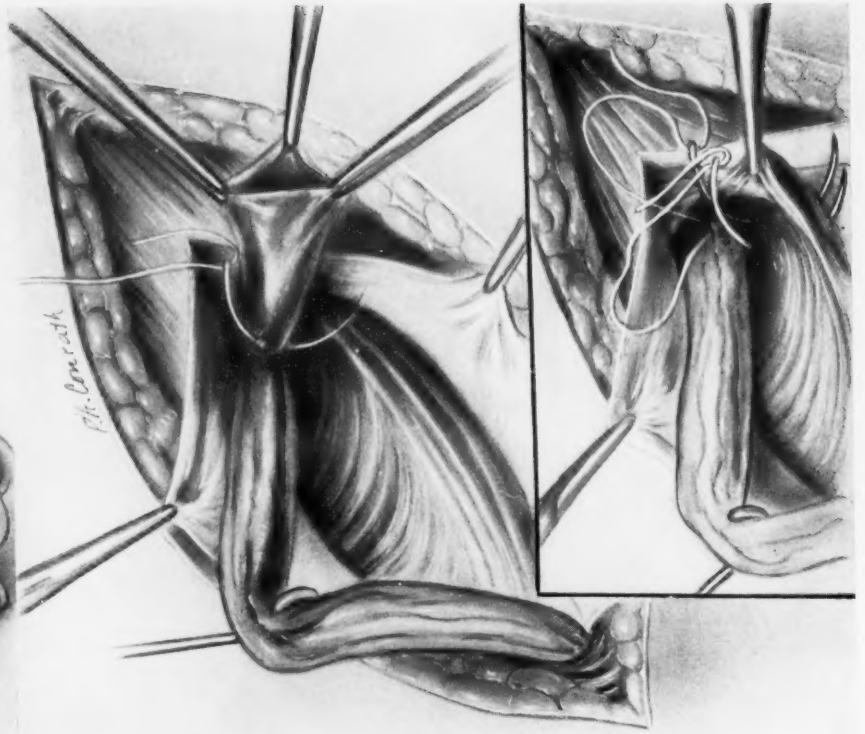


FIG. 2.—Showing lateral retraction of elements of cord, and transfixion of neck of sac. (Inset)—Method of dislocating neck of sac inward.

surrounding structures by gauze dissection. After it is free from the ring, it is transfixed, ligated and excised. The stump is carried inward and downward under the muscle and anchored by mattress suture, thus carrying the peritoneal dimple away from the posterior canal wall and anchoring it near enough the bladder to prevent a pull. As we believe the posterior wall is distensible and unstable in its entire length, we do not disturb but reenforce it in the following manner with double No. 1 20-day catgut: The first suture starting from without inward through the fascia lata, just below Poupart's ligament as closely as possible to the pubic spine grasping the conjoined tendon and returning from within outward through or just above Poupart's ligament where it is tied, leaving the knot external to the fascia. Each staggered suture is placed after the same manner three-eighths of an inch apart ex-

FIG. 3.—Showing technic of suture of conjoined tendon to Poupart's ligament, with knots external to the fascia.

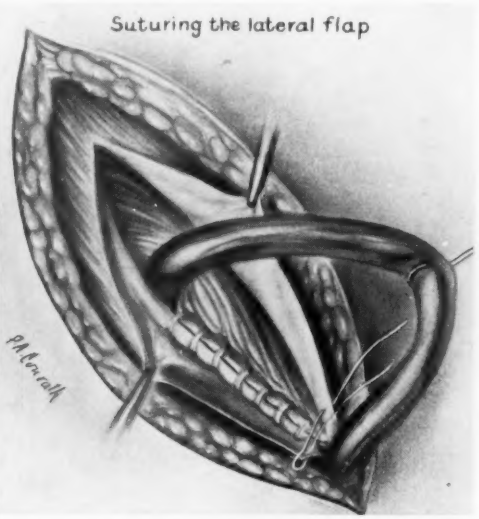


FIG. 4.—Method of imbrication of the aponeurosis of the external oblique.

tending up the border of the rectus sheath as high as is necessary to cover the posterior canal wall, raising the cord a bit higher and giving it more angulation than formerly, closing the abdominal ring to the proper size. The elasticity of the muscle above the ring prevents strangulation of the cord. The lateral flap of external oblique aponeurosis is carried medially as far as possible and sutured to the deep side of the medial flap with a suture exactly similar to the first row, leaving the knots external to the fascia. The medial flap is now carried laterally and sutured either to Poupart's ligament or to the fascia lata. The skin and superficial fat are sutured with interrupted silkworm gut sutures about one inch apart, the needle entering the skin one-half inch from the margin and extending straight through fat. Above the internal ring the skin suture includes the fascia, thus obliterating space. The friction or surgeon's knot of three turns is used

to prevent puckering to secure proper approximation of the skin and to insure a flat comfortable knot.

We believe this method gives us an ideal posterior wall; that the arching of the internal oblique and transversalis above the abdominal ring gives us a perfect valve or sphincter and that the external ring which has been destroyed plays no part in the formation of hernia. Trauma to the transplanted cord has been mentioned; however, when we consider the flexibility of the abdominal wall, it is quite apparent that the only possible point of trauma is as it passes over the pubic arch, which region has not been disturbed. Our experience with reference to recurrence warrants our belief that the procedure is eminently satisfactory. The fact that we have for

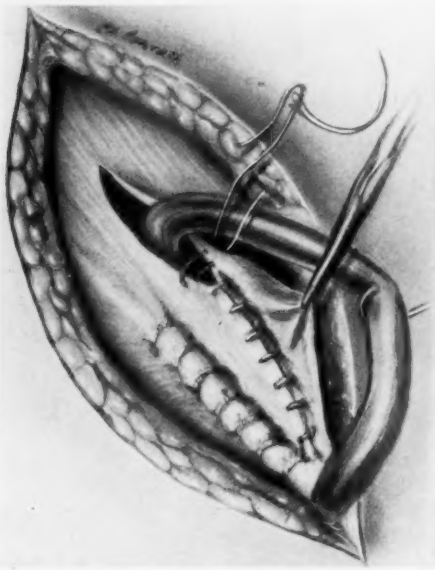


FIG. 5.—Extent to which imbrication in Fig. 4 is carried.

FIG. 6.—Situation of final tacking sutures in the medial flap of the external oblique aponeurosis.

many years been connected with the Hospital Department of a large railway where hernia is especially common, and where the patients can return for operation because of recurrence without expense, leads us to believe that we would see more of our failures than the majority of surgeons.

This operation was originally reported before the Southern Medical Association in 1925, since which time we have personally performed more than 500 operations with but two recurrences. The first occurred following a postoperative pneumonia. The second recurrence followed operating upon a hernia, in which the entire posterior wall protruded well down under Poupart's ligament, which was found to be practically absent and fascia lata so thin as to be of little value. The patient was advised that recurrence was probable. This eventuated after three months. At time of reoperation so much connective tissue had been borrowed by Poupart's liga-

ment from the rectus sheath that the ligament was practically of normal density, and substantial repair was effected.

I have operated upon several recurrences during the past ten years, in cases originally performed by my assistants. In each instance the recurrence was at the abdominal ring. There is no question in my mind that the first suture line was not carried high enough in these instances.

The injection treatment of hernia originated with Velpeau 100 years ago. He used iodine in the attempt to obliterate the hernial sac. In Germany, Schwalbe in 1887 used alcohol, and it is unfortunately still used by many European physicians. Some years later the paraffin method was introduced and used for quite some time, finally being discarded by reputable surgeons, but it is still employed by quacks and charlatans. In the last five years much experimental work has been done with the injection method. It is claimed that the injection causes a proliferation of connective tissue which blocks the canal and prevents protrusion. Quite a number of different preparations have been used. In the advertising of one the statement is made that the technic of the injection method is easily acquired. In another, that it is not necessary that the treatment be administered by a surgeon. The talking point that the patient remains ambulatory, loses no time from his work and does not require hospitalization, has popularized the treatment both with the laity and the insurance companies. Regardless of the economical feature any patient would grasp at such a method rather than undergo a major operation. Only when a large number of surgeons use a certain method over a sufficient period of time can the true merit of the procedure be ascertained. I have had no experience with the injection procedure and only wish to warn of the possibility that this method might be taken up by quacks and medical cults, resulting in a great deal more harm than good.

CONCLUSIONS

(1) A congenital weak posterior inguinal canal wall should be considered responsible for inguinal hernia.

(2) The operative procedure detailed both removes the sac and reinforces the posterior canal wall, to a degree in considerable excess of the normal, as insurance against recurrence.

(3) The enthusiasts of the injection method possibly exaggerate its advantages and minimize the results of operation.

REFERENCES

[1] Colcord: International Jour. Surg., 1919.
[2] Brown: ANNALS OF SURGERY, 1904.
[3] Russell: Lancet, 1900.
[4] Pitzman: ANNALS OF SURGERY, 1921.
[5] Andrews: ANNALS OF SURGERY, 1924.
[6] McArthur: J.A.M.A., November, 1901.
[7] Seelig: Am. Surg. Assn., 1924.

DISCUSSION.—DR. BRADLEY L. COLEY (New York, N. Y.).—Doctor Woolsey has referred to the large number of operations that have been proposed for inguinal hernia, which means that no one method is satisfactory and applicable to all types of hernia. In the small oblique hernia in young adults we feel that the Bassini operation is applicable and in our experience has given satisfactory results.

Dr. Carl G. Burdick, with whom I am associated at the Hospital for Ruptured and Crippled, began about eight years ago to divide the cord completely in large, neglected, scrotal herniae in men beyond middle life, and in a recent report gives the results in 200 such cases. Having divided the cord, the stump is allowed to retract into the abdomen, which permits of complete closure of the opening and thus eliminates a potential weak spot in the repair.

The indications for this radical procedure have been extended to include large sliding herniae, large scrotal herniae in the aged, which are either irreducible or cannot be satisfactorily retained by a truss, recurrent herniae in individuals over 50 with a normal testicle on the opposite side, where there have been one or more unsuccessful attempts at a radical cure, and finally recurrent herniae in younger subjects, who by reason of their recurrences are incapacitated from performing their occupations and where a perfectly normal testicle exists on the opposite side.

Direct hernia is a different problem, and here the traditional Bassini operation is apt to be followed by a high percentage of recurrences. For more than a decade we looked upon the repair by fascial sutures as the most satisfactory method of dealing with direct inguinal hernia. However, recently a careful study of end-results in approximately 1,200 such fascial suture operations revealed results which were far from realizing our hopes and expectations, so that in the last year or two we are performing this operation much less frequently.

The use of silk for ligatures and sutures was undertaken with some misgivings, as our policy in the past always tended to avoid non-absorbable material at all times. During the past two years, however, the silk technic has steadily displaced the use of other suture materials, and we are convinced that by employing it we have greatly lowered our percentage of infection and improved the operative results. Whether this can be attributed solely to the use of the silk or to the meticulous care in handling the tissues and securing absolute hemostasis and accurate coaptation, which is an integral part of the silk technic, I do not know.

Finally, a word about the injection treatment of hernia. About a year ago we felt that this method should be investigated, because of the voluminous reports of its successful employment in the hands of others. Accordingly, in November, 1934, we commenced to use the injection method, and employed it in some 50 cases. Several injection fluids were tried, including Pina Mestre, Galtanol, Proliferol and O'Malley's Solutions. The immediate results during and following the course of injections in many instances were most encouraging and for a time we were quite enthusiastic. Following the removal of the truss and with the lapse of time, in a high percentage of cases which at first seemed to be controlled, it was found on subsequent follow up examination that the hernia was still present. The uncertainty of the method, the prolonged period of treatment and observation, the necessity for wearing a large uncomfortable truss night and day for a prolonged period, and the occasional development of a hydrocele following the injections prompted us to discard it almost entirely.

Dr. Adrian S. Taylor (Clifton Springs, N. Y.).—I investigated all hernia operations done in the Johns Hopkins Hospital, with regard to recurrence, in the cases in which operation had been done in Doctor Halsted's clinic during the second 20 years of its existence. Doctor Bloodgood had reported the results during the first 20 and, strange to say, I found exactly the same results. In indirect hernia, the average time of recurrence in a large series was 48 months after operation, while in direct hernia, recurrence was found to occur within the first year. In all operations for hernia there was an average recurrence of 5.6 per cent in indirect cases, and 18 per cent in direct. I agree thoroughly with the essayist that an important cause of recurrence, in all cases of hernia, is to be found in a weak posterior inguinal canal wall. Doctor Halsted very early pointed out a method of strengthening this wall which obeys all the requirements for plastic surgery. I refer to his method of drawing the cremasteric muscle and fascia up underneath the conjoined tendon, as the first layer in the closure. By so doing the transversalis fascia is also strengthened.

I am delighted to hear my friend from New York speak of the use of silk. I wish to refer to a sentence in an article by Cushing, which appeared in Science after the death of Doctor Halsted, in which he said, "He showed how silk could be safely buried in the tissues, an important principle many surgeons are incapable of learning." Now, after so many years, there is an increasing appreciation of the exquisite technic taught by Doctor Halsted, which is perhaps dependent upon the use of a fine, easily sterilizable suture material.

Doctor Woolsey has said that infection produces fibrosis. During the first year of the Johns Hopkins Hospital six hernia operations were performed by Doctor Halsted's resident, who used the old McBurney technic, in which the sac was tied off and the wound packed with gauze and allowed to heal by granulation. In these cases there was prompt recurrence in 50 per cent. The same high percentage of recurrence was found in those cases in which infection occurred. Here, of course, fibrosis was extreme. In the injection treatment, fibrosis must be depended upon, and if temporary cure be attained, the incident of subsequent recurrence must be high.

Dr. George A. Hendon (Louisville, Ky.).—I have always approached the problem of hernia on the principle of a plastic operation, keeping prominently in view certain underlying principles, upon which success depends: (1) The extent of denuded areas that are in apposition. (2) The amount of tension that is necessary to secure the contact. In order to meet with the first requirement of bringing broad surfaces together, mattress sutures of kangaroo tendon are carried through the conjoined tendon and aponeurosis of the external oblique jumping over the canal and including Poupart's ligament and the adjacent fascia. The position of the cord is not disturbed. When the sutures are tied, fascial inversion with broad contact is secured. I think that by emphasizing this plastic principle we add a great deal to the security of the operation. It applies in all forms of hernia.

Dr. John T. Moore (Houston, Tex.).—I have been highly instructed by the dissertations on hysterectomy and hernia, but where have we gotten as a group? Each man has presented something worth while. I have always looked upon this Association as a great clearing house for surgery, to have it presented and approved so that the mass of those doing surgery in the country could profit as we have done as a group. To take a lesson from the oil people: they have had the problem down in our country of

drilling through heaving shale. They have found a method that was successful, because their engineers have now compared their work and have gotten together. In other words, by comparing methods, a standard of operations puts them through heaving shale by the best known methods. Is it not possible that this organization, the American Surgical, and other Associations could work out a plan whereby an hysterectomy, total or subtotal, or hernia operation, in this way or that way, could be done by a somewhat standardized technic—one accepted by the best surgeons?

I am also glad to know that many men outside of Johns Hopkins have learned that fine silk, in clean wounds, is one of the best suture materials we have ever had. Still, we go all around the country and see our surgeons talking about a lot of things that it seems to me have been settled for a long time.

Dr. W. D. Wise (Baltimore, Md.).—I was interested in this paper on hernia because we have had a feeling for a long time that fascia to fascia repair is a great improvement over the results of sewing muscle to fascia, although that is done also, because to bring down the internal oblique and the transversalis to Poupart's ligament is the logical thing to do. However, the plan presented by the essayist gives a fascia to fascia approximation that I think will, in most instances, do away with the necessity of the Gallie operation or similar procedures.

The operation we have been performing since 1919 is quite similar to that of the essayist. We, however, carry our sutures a little higher under the cord, making the cord point more obliquely, which increases the valve effect. I would suggest to Doctor Woolsey that, as he carries his suture through Poupart's ligament, he go down and pick up the pectineus fascia, thus helping to prevent a femoral hernia. In the repair of inguinal hernia there is sometimes a tendency to open up the femoral canal. Several years ago I demonstrated a suture intended to prevent this.

In some cases when the sac has been dissected down to the deep ring and one reaches the point where an attempt has been made by nature to close it but has not succeeded, if the finger is put through the neck of the sac and lifted slightly, one will find another potential sac there, something like a saddle-bag hernia but lateral to the inferior epigastric artery. I do not see why this would not be an element in a certain number of recurrences of hernia. This can easily be removed along with the sac. I always look for it and in quite a percentage of cases it is found and removed.

Dr. R. A. Woolsey (closing).—I have very little to say, other than that on one occasion many years ago in Chicago at Dr. Albert Ochsner's Clinic, I heard him comment on a recent article on hernia written by a New York surgeon quoting his percentage of recurrence. He remarked that this man had very little chance of knowing of the majority of his recurrences, since they went to other surgeons for operation, and that he himself had quite recently operated upon four of the author's recurrences.

I believe the most important step in a hernia operation is the fascia to fascia imbrication as set forth in this procedure.

SURGICAL TREATMENT OF GIANT CELL TUMOR

BRADLEY L. COLEY, M.D., AND NORMAN L. HIGINBOTHAM, M.D.
NEW YORK, N. Y.

FROM THE BONE TUMOR DEPARTMENT OF THE MEMORIAL HOSPITAL, AND THE HOSPITAL FOR RUPTURED AND CRIPPLED, NEW YORK, N. Y.

AN HISTORIC study of giant cell tumor of bone shows that the treatment of this condition has followed closely discoveries in the fields of pathology, roentgenology and radiology. Among those who have contributed prominently to our knowledge are: Astley Cooper, Lebert, Paget, Nelaton, Virchow, Koenig, Hinds, Bloodgood, W. B. Coley, Ewing, Codman and others.

Before the beginning of the present century, amputation was the usual treatment since these cases were seldom distinguished from the malignant bone sarcomata. With the advent of roentgenology, however, the distinctive appearance of the giant cell tumor, together with the characteristic microscopic features, made it possible to recognize the process as a distinct entity. Bloodgood (1910) and W. B. Coley (1907) were the first of the present day writers to advocate conservative treatment, warning against amputation as an unnecessarily radical procedure. One way in which Bloodgood accomplished his purpose was to insist upon dropping the term giant cell sarcoma and substituting that of benign giant cell tumor. The method he advocated was: curettage and cauterization with an escharotic, such as phenol. Curettage of the benign giant cell tumor was probably first performed by Koenig, about 1894. Hinds (1895) first combined curettage with chemical cauterization of the cavity and established this method as a curative measure. His case was traced and known to be well 26 years later. Bloodgood, in 1923, gives his own experience with curettage and cites cases so treated by more than a dozen other American surgeons.

Irradiation therapy came into general use about two decades ago in the treatment of all forms of malignant disease, so it was natural enough that it should be tried for giant cell tumor. Herendeen was among the first to demonstrate its effectiveness and to show that cures can be obtained in many of these tumors. Other roentgenologists quickly followed the lead, and now the treatment by roentgen ray has, in some localities, largely superseded surgical measures.

It is admitted that surgical treatment of a few decades ago, while often successful, not infrequently resulted in infections, sinus formation and fungation of tumor tissue which occasionally necessitated an amputation. The chief reasons for these bad results were threefold, *viz.*: (1) An exploratory operation was often done first with removal of only sufficient tumor tissue for biopsy, or the operator failed to remove all tumor tissue by curettage because exposure was often inadequate, and hemorrhage frequently so alarming as to cause him to desist before achieving complete extirpation. (2) The practice of packing the bone cavity with gauze frequently led to secon-

dary infection and fungating wounds, for when, to an inadequately removed giant cell tumor, secondary infection is added, the result is quite likely to be eventual loss of limb. (3) Failure to use chemical cauterization after curettage is another explanation of recurrences, because cauterization of the cavity wall destroys any small remaining traces of tumor tissue.

We shall attempt to show why we believe properly performed surgical treatment gives superior results in accessible tumors of the long bones, and in particular, of the weight bearing long bones.

Fortunately, the surgical picture has changed. About ten years ago one of us (Coley) suggested the importance of immediate closure of the wound without drainage or packing after curettage for giant cell tumor. Striking improvement in end-results was noted, and the procedure soon became routine on the Bone Tumor Services at the Memorial Hospital and at the Ruptured and Crippled Hospital. Further technical changes included the use of a large enough window to permit thorough curettage of the entire cavity under direct vision; rapid removal of tumor tissue and the use of a tourniquet to minimize hemorrhage and to prevent obscuring the field during the curettage. These measures were reinforced by a most rigid preoperative skin preparation.

That the method has yielded satisfactory results is shown by the following. In the last 21 operations upon 20 consecutive cases treated surgically, wound healing by first intention resulted in every instance with the exception of one recent case with an infected sinus, from operations performed elsewhere, in which we were unable to obtain primary closure of the wound. Four of these cases were recurrent when operated upon, the primary operation having been done elsewhere. A fifth recurred after the first curettage, but is apparently cured by the second. With the exception of one recurrent case that showed evidence of malignant degeneration after a second curettage and died of pulmonary metastases following amputation, all of the remaining 19 cases are now well, have good function of the adjacent joint and are free from symptoms. In each instance repair is demonstrated by roentgenogram to be adequate. In only one case was there evidence of pathologic fracture after curettage; this patient was hurt in an automobile accident.

BRIEF REPORTS OF 20 CONSECUTIVE CASES TREATED SURGICALLY

Case 1.—D. C., male, aged 39. Admitted to Memorial Hospital July 8, 1931 with a history of slight swelling in the right knee and tenderness over the inner condyle of the femur. Roentgenograms showed a giant cell tumor of the lower end of the right femur and aspiration biopsy supported the diagnosis. Operation July 15, 1931. The diseased area was curetted and cauterized and a circular plaster case applied. The splint was removed two months later and a walking Thomas caliper worn for four months. Since then he has been able to get about without any support, is symptom free and has full function of the knee. Last seen September 25, 1935, more than four years after operation.

Registry No. 1305: Giant Cell Tumor.

Case 2.—W. P., male, aged 26. Admitted to Memorial Hospital August 6, 1931, with a history of pain and disability in the left knee of two years' duration. Roentgenograms

showed a giant cell tumor of the upper end of the left tibia. Operation September 3, 1931. The diseased area curetted and cauterized. The wound was closed above a small strip of gauze packing at the lower angle. Two roentgen ray treatments had been given the previous week (500 *r* units medial and lateral). The leg was kept in a plaster case for six weeks and Coley's toxins administered. He was able to walk without support two months after operation, returned to work as a chauffeur three months after operation and has shown normal range of motion since. Last seen April 17, 1935, more than three and one-half years after operation.

Registry No. 1303: Giant Cell Tumor.

Case 3.—G. O., male, aged 44. Admitted to Memorial Hospital July 1, 1931. He had been treated in July, 1930, for a fracture of the femur sustained from a fall. He refractured his femur in November, 1930, when his crutch slipped. In April, 1931, after another fracture, a cyst was diagnosed and he was given ten roentgen ray treatments without improvement. On admission to Memorial Hospital the roentgenograms showed an extensive giant cell tumor of the lower end of the left femur. Operation July 15, 1931. The diseased area was curetted and cauterized and the limb supported in a plaster case for two and one-half months. Eleven injections of Coley's toxins were given. Weight bearing was begun six weeks later. He was able to walk without support seven months after operation. Since then he has had a range of motion from 180° extension to 90° flexion, with no symptoms. Last seen September 30, 1935, more than four years after operation.

Case 4.—E. B., female, aged 31. Admitted to Memorial Hospital June 10, 1933, with a history of fracture above the left knee sustained three years previously and resulting in 4 cm. shortening and disability. Roentgenograms showed a giant cell tumor of the lower end of the left femur. Operation June 26, 1933. The diseased area curetted and cauterized. A plaster case was applied for three weeks; then a walking Thomas caliper for six months. At last examination, September 4, 1935, she had a one-quarter inch raise on her heel, walked with a barely perceptible limp and showed a range of motion at the knee from 180° extension to 85° flexion.

Registry No. 1539: Giant Cell Tumor.

Case 5.—H. G., male, aged 22. Admitted to Memorial Hospital May 25, 1931, with pain in the knee of six months' duration. Roentgenograms showed a large giant cell tumor in the upper end of the left tibia. Operation May 27, 1931. The diseased area was curetted and cauterized. He was discharged from the hospital in two weeks and could walk without support and without a limp three weeks after operation. He showed no limp or deformity and could extend to 180° and flex to 90° when last seen, November 6, 1935, four and one-half years after operation.

Registry No. 1296: Giant Cell Tumor.

Case 6.—M. L., male, aged 44. Admitted to Memorial Hospital November 8, 1933, after operation at another hospital in August, 1933, for giant cell tumor of the upper end of the right tibia. As symptoms persisted he was again operated upon November 11, 1933, curettage and cauterization being performed. Giant cell tumor was reported by the pathologist. Symptoms of pain persisted and an aspiration biopsy was performed which revealed a malignant tumor. The leg was amputated January 6, 1934, and a tumor thrombus found in the femoral vein. Microscopic sections showed malignant sarcoma. There was an early recurrence in the stump and pulmonary metastases by May 16, 1934. Patient died July 20, 1934, one year after his first operation.

Registry No. 1697: Giant Cell Tumor.

Case 7.—T. B., male, aged 15. Admitted to Memorial Hospital October 3, 1930, after curettage and cauterization of a giant cell tumor of the lower end of the right femur at another hospital in July, 1930. He had had pain in the right knee for one month prior to operation. He was given a course of Coley's toxins and the plaster case removed three

months after operation. Since then he has been symptom free and has full function of his knee. Last seen August 7, 1935, more than five years after operation.

Registry No. 1698: Giant Cell Tumor (malignant).

Case 8.—A. W., male, aged 40. Admitted to Memorial Hospital May 9, 1930, with a history of "rheumatism" for 13 weeks. Roentgenograms showed giant cell tumor of the upper end of the right tibia. He was given three cycles of roentgen ray therapy, using two ports, in May, June and the following January. Operation February 25, 1931. Curettage and cauterization were performed and were followed by primary wound healing. He was discharged from the hospital in six weeks and had return of full function of the knee in four months. Last seen April 17, 1935, with full function and symptom free, more than four years after operation.

Registry No. 1382: Giant Cell Tumor.

Case 9.—M. D., female, aged 17. Admitted to Memorial Hospital June 19, 1935. Roentgenograms showed giant cell tumor of the upper end of the right tibia of the epiphyseal chondromatous type of Codman. Operation June 26, 1935. Curettage and cauterization were performed and followed by primary union. She walked well without crutches in two and one-half months. At last examination, November 27, 1935, she could flex the knee to 85° and extend it to 175°, and walked without any limp four and one-half months after operation.

In process of registration.

Case 10.—W. M., male, aged 30. Admitted to Memorial Hospital July 14, 1935, following three operations for giant cell tumor of the upper end of the left tibia. There was a draining sinus at the time of admission, but as the tumor had evidently been incompletely removed, operation (curettage and cauterization) was performed July 24, 1935. The pathologic report was aggressive giant cell tumor. Primary wound closure was attempted under tension, but the wound separated and left a discharging sinus. He last reported (October 8, 1935) that the wound was still draining.

Case 11.—J. L., female, aged 11. Admitted to the Ruptured and Crippled Hospital October 12, 1931, with a history of pain and swelling of the right knee for six weeks. Roentgenograms showed giant cell tumor of the upper end of the right tibia. Curettage and the cavity packed by another surgeon October 19, 1931. The tumor recurred and on February 1, 1932, a second operation was performed—curettage and cauterization. This was followed by a series of Coley's toxins. There was normal function of the knee in two months and she was symptom free with full function of the knee when last examined, February 25, 1935.

In process of registration.

Case 12.—L. L., female, aged 13. Admitted to the Ruptured and Crippled Hospital November 4, 1932, with a history of pain and swelling in the right wrist of ten months' duration. Roentgenograms showed a giant cell tumor of the lower end of the right ulna. On November 7, 1932, curettage and cauterization were performed and the wound healed per primam. This was followed by a series of Coley's toxins. When examined a year later she was symptom free and had full function of her wrist.

Registry No. 1706: Giant Cell Tumor.

Case 13.—E. B., male, aged 41. Admitted to the Memorial Hospital January 18, 1929, following curettage at another hospital. The wound had been packed and was infected. It required five months to sterilize the cavity sufficiently to skin graft, after which healing was complete and the leg was in excellent condition. He moved out of the city, but reports frequently by letter that his knee is as good as the other one. His last letter was August 22, 1935, nearly seven years after operation.

Registry No. 1086: Giant Cell Tumor.

Case 14.—R. P., male, aged 37. Admitted to the Ruptured and Crippled Hospital April 18, 1928. He had had an amputation of the left leg November 30, 1920, presumably

for osteomyelitis. He had had pain in his right knee for five months prior to admission to the hospital. Operation April 20, 1928. Curettage and cauterization. He was given a series of Coley's toxins and was then transferred to the Memorial Hospital for radium treatment. He was given 32,000 mg.h. with the radium element pack at 10 cm. distance in June, 1928. He has remained symptom free and was reported in good condition on December 6, 1934, more than six and one-half years after operation.

Registry No. 896: Giant Cell Tumor.

Case 15.—J. S., male, aged 29. Admitted to Bellevue Hospital April 13, 1935, with a history of pain in the right hip of six months' duration. Roentgenograms revealed giant cell tumor of the upper end of the right femur and on April 20, 1935, curettage and cauterization were performed and a plaster spica applied. The spica was removed four months later and the patient fitted with a walking Thomas caliper. He was seen November 22, 1935, the wound had healed per primam and there was good range of motion at the hip. Roentgenograms showed good calcification in the cavity, but because of the size of the original tumor the patient was advised to continue to use his caliper splint.

Case 16.—R. L., female, aged 26. Admitted to the Ruptured and Crippled Hospital September 18, 1934, with a history of pain in the right knee (diagnosed giant cell tumor and operated upon one year ago at another hospital). She had recently fallen and injured the affected knee and on admission roentgenograms showed a pathologic fracture with depression of the medial condyle of the tibia. Traction was tried; then a plaster encasement; but symptoms persisted. Operation, November 26, 1934. The involved area in the upper end of the right tibia was curetted and cauterized. The wound healed by primary union and a series of Coley's toxins was given during convalescence. A plaster case was applied for three months; then a walking Thomas caliper, which she still wears. When last examined (November 27, 1935) patient was symptom free and the range of motion at the knee was from 180° extension to 135° flexion.

In process of registration.

Case 17.—P. O., male, aged 38. Admitted to the Ruptured and Crippled Hospital July 5, 1932, with a history of pain in the left knee of three months' duration. Roentgenograms revealed a giant cell tumor of the lower end of the left femur. Operation, July 11, 1932. The involved area was curetted and cauterized, and in four months he was able to walk without support and was symptom free. Following a local injury his symptoms returned and the area was again curetted and cauterized February 18, 1933. He was then given a series of Coley's toxins. He remained well with good function of the knee until September 18, 1934, when he fractured his femur through the involved area. He was treated in Russell traction for two weeks and then wore a plaster spica for two months. He then used a walking Thomas caliper for six months. When last seen (December 4, 1935) he was symptom free, had good function of the knee, and roentgenograms showed good bone regeneration in the cavity.

In process of registration.

Case 18.—A. S., male, aged 16. Admitted to the Ruptured and Crippled Hospital June 22, 1930, with a history of pain and swelling of the right ankle of 12 months' duration. He had been operated upon at another hospital in January, 1930, but his symptoms had recurred. Roentgenograms revealed a giant cell tumor of the lower end of the right tibia—recurrent. Operation, June 24, 1930. Curettage and cauterization, the wound healing by primary union. December 7, 1935: patient free of symptoms; has perfect function of ankle.

Case 19.—C. K., male, aged 23. Admitted to Lincoln Hospital March 19, 1935, with a history of swelling in the right ankle following a kick. Roentgenograms revealed a large area of cystic degeneration in the lower portion of the right tibia. Operation, March 23, 1935. Curettage and cauterization were performed, the wound healing by primary union. Pathologic Report: Benign giant cell tumor. December 3, 1935: wound

is well healed. The external appearance of the ankle is almost normal and the motions of the ankle are normal in all directions. There are no symptoms. Result functionally, anatomically and economically excellent.

In process of registration.

Case 20.—J. L., male, aged 15. Admitted to Lincoln Hospital March 30, 1935, because of pain and swelling on the dorsum of the left foot. History of injury to foot against a bench four months prior to admission. Examination showed a swelling over the dorsum of the left foot at the base of the second and third toes. Roentgenograms revealed a large cystic swelling, expanding the distal two-thirds of the third metatarsal. Operation, April 1, 1935. Resection through the base of the third metatarsal bone with removal of tumor mass, the wound healing by primary union. Examination (December 4, 1935) revealed that the wound was well healed, and that patient walked without limp. Wore foot plate for support of transverse arch. Result satisfactory.

In process of registration.

Indications for Surgical Treatment.—In general, it may be stated that giant cell tumors occurring in bones inaccessible to surgical approach should be treated by irradiation. Tumors of the ulna and fibula, if quite extensive, are amenable to resection, without great sacrifice of function, and with assurance of successful end-result. Tumors of the upper end of the femur are not always readily accessible to curettage and the operation may be quite difficult; irradiation may therefore have to be employed in this location. Tumors occurring in the lower femur and upper tibia seem to us to be particularly adapted to curettage. They are accessible, the functional result is excellent, the period of disability is shortened with obvious economic advantages, and pathologic fracture is avoided. We find that pathologic fracture has complicated approximately one-fourth of our giant cell tumors of the long bones treated by irradiation.[1] This high incidence doubtless could be cut down by greater restriction of activity and more careful splinting and protection during the prolonged period often necessary for bone regeneration following roentgen ray therapy. But it has been our experience that where pathologic fracture has occurred, joint function usually suffers permanent impairment.

The effect of therapeutic doses of roentgen ray on joint structures, especially synovial membrane, but also muscle and fascia, has never, so far as we know, been studied experimentally. Clinically, however, we have seen a number of cases in which irradiation has produced obvious permanent damage to skin and underlying soft tissues. No doubt the dosage has been modified in more recent years so as to render these sequelae less frequent. Yet the amount of irradiation required to control the giant cell tumor has never been established and no doubt varies with individual tumors and probably is influenced by the age of the patient, rate of growth of the tumor, location in bone and especially its vascularity. Dosage is still a matter upon which wide variation of opinion exists, but it seems safe to say that doses formerly employed were generally excessive and too often repeated. It is certainly wiser to err on the side of under treatment. Excessive doses

may completely inhibit bone regeneration and even lead to eventual amputation.

Selection of Type of Treatment.—It is apparent that roentgenologists as a rule prefer irradiation, while surgeons favor curettage. Pathologists, who are more apt to see the unfavorable results of cases treated by both methods, are divided in their opinions. It is our conviction that unless one has had an opportunity to see a considerable group of cases treated by both methods,

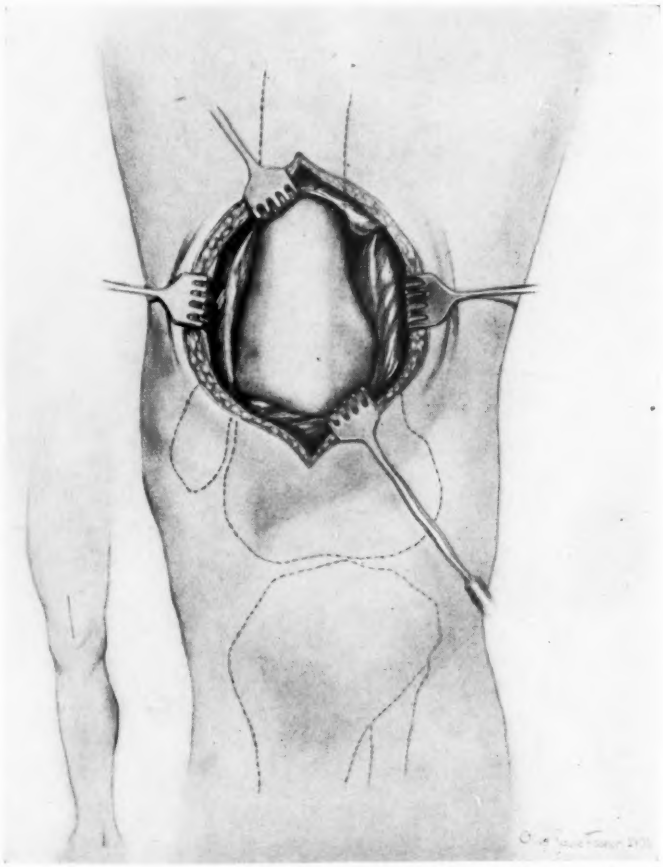


FIG. 1.—Shows exposure of involved portion of the femur with retraction of, rather than division of, the muscles.

singly and in combination, to follow them for years and to treat the complications and sequelae that arise, he is scarcely in a position to decide upon their merits.

Operative Technic.—Having decided that a given case is suitable for operation, our procedure is as follows. The skin is prepared as painstakingly as for any open bone operation; we prefer a 48 hour preparation. Provision is made for transfusion immediately after operation, in case undue loss of blood creates the necessity for it. Under general anesthesia (gas-oxygen or avertin) and using a tourniquet the skin incision is made over

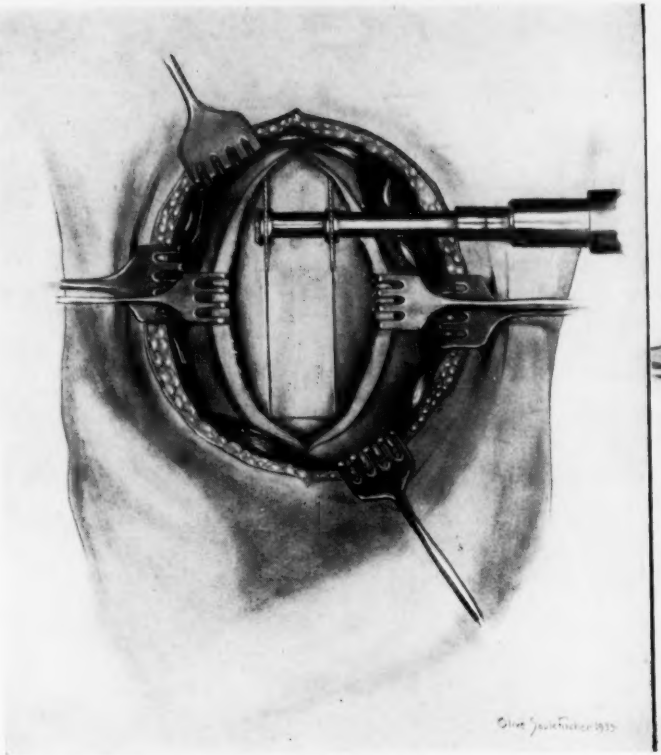


FIG. 2.—Shows periosteum divided and retracted and window of cortical bone outlined by motor saw. This step can be done with an osteotome and, where the shell is very thin, sometimes by knife or heavy scissors.

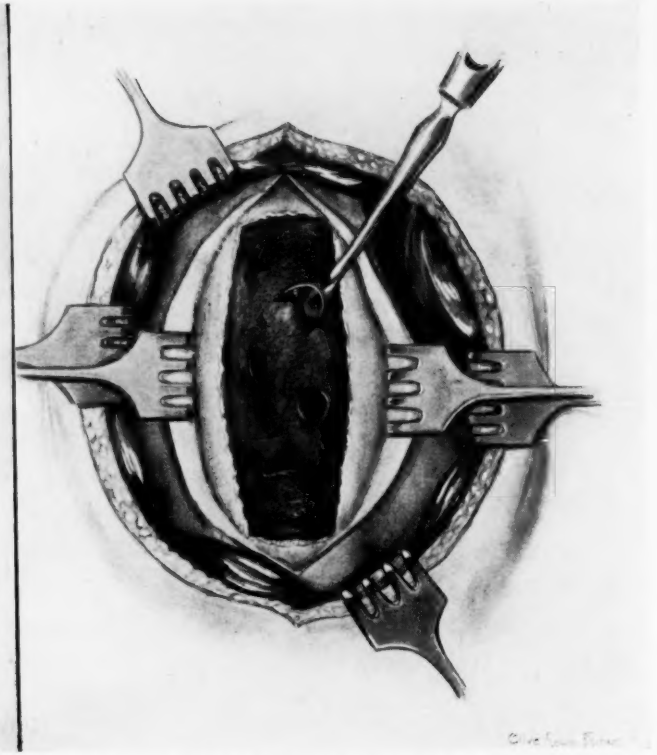


FIG. 3.—Shows window enlarged by rongeur and curettage of tumor tissue being carried out.

the tumor bearing portion of the bone. The selection of the exact site of the incision and care in avoiding the neighboring joint cavity are matters of importance.

The incision passes down to periosteum through fascia and muscle, splitting rather than dividing the latter whenever possible (Fig. 1). The periosteum is divided and stripped back to expose the area of cortex which is to be removed for access to the tumor. A rectangular window is then made by an osteotome or twin-bladed motor saw (Fig. 2). The window is then enlarged with a rongeur until a thorough curettage can be performed under direct vision, aided by palpation (Fig. 3). No unnecessary sacrifice of cortical bone should be made. To assist in getting at the roof of the

FIG. 4.—Instruments useful in the surgical treatment of giant cell tumor.

cavity and to insure complete removal of all particles of tumor tissue and cancellous bone, it is helpful to use a curet with an angular handle (Fig. 4).

Tumor tissue is thus rapidly, but carefully, curetted and the cavity repeatedly flooded with Dakin's solution or normal saline solution. When the surface of the bone cavity is clean, the interior is thoroughly swabbed out with a saturated solution of zinc chloride (or, if this is not available, with pure phenol followed by alcohol), which is allowed to remain in contact with the bone for about one minute, care being taken to protect the soft parts from contact with the escharotic solution, which is then flushed away by flooding the cavity with Dakin's solution. Periosteum, muscle, fascia and skin are closed in separate layers with interrupted sutures without drainage.

We do not favor the stuffing of the cavity with bone chips, for the bone shell usually possesses sufficient regenerative power to restore continuity. Fine silk is used for the skin, with care to maintain accurate approximation. The tourniquet is removed and a bulky gauze dressing with sterile sheet wadding and snug flannel bandage is applied. Plaster encasements or splints are used where indicated. For the first 72 hours after operation the part is kept elevated. When the skin sutures are removed, the wound is redressed, still maintaining moderate pressure as at the initial dressing. Great care is taken to prevent a pathologic fracture. Thus in lesions of weight bearing bones the use of caliper splints is indicated until roentgenologic evidence of adequate bone regeneration is apparent. In some cases the slight degree of involvement permits a very brief period of restraint; in others some months are required to fill up the bone defect sufficiently to permit full function with safety. But during the interval attention is paid to restoring muscle tone and range of motion of neighboring joints, at the same time guarding against fracture.

Treatment of Malignant Giant Cell Tumor.—Whether one believes that malignant giant cell tumor is actually malignant from the beginning and merely simulates the benign variety, or that it represents a transformation from a benign process into a malignant one, does not alter the fact that once this condition is recognized, treatment by radical surgery alone offers hope of saving the life of the patient. For metastasis to the lungs is the usual termination.

It is our belief, however, that malignant transformation of a benign giant cell tumor, though an uncommon condition, is favored by chronicity and enhanced by incomplete surgical removal, particularly when combined with irradiation. For reasons not clearly understood, curettage and irradiation do not form a desirable combination in the treatment of giant cell tumor.

Though we have seen malignant degeneration follow treatment by surgery alone and by irradiation alone, it occurred more often where a combination of these methods was employed. There is some evidence, therefore, to suggest that irradiation is a factor in the occasional transformation of these benign tumors into definitely malignant ones. The application of radium into a bone cavity is mentioned only to be condemned. We believe that any giant cell tumor, that has not been successfully treated, is a potential source of danger of malignant transformation.

Age of the patient has been a factor to which too little attention has been paid. We find that in patients in the late thirties and early forties the giant cell tumor runs a more aggressive course, requires a more thorough extirpation by curettage, tends to respond less consistently to irradiation, and may become malignant. This is due no doubt to the decline in reparative power of cortical bone after the age of 20. The prognosis is therefore more uncertain in these older patients.

SUMMARY.—Our experience with both the surgical and the roentgen-

ologic treatment of giant cell tumor is based on a study of 90 cases at the Memorial Hospital, the majority of which were treated by irradiation, and in addition, a considerable number of cases treated at the Hospital for Ruptured and Crippled, Bellevue Hospital and Lincoln Hospital. In brief, we consider that the treatment of giant cell tumor of bone is neither wholly surgical nor roentgenologic. Tumors occurring in the spine, pelvis and other inaccessible locations are best treated by irradiation. Tumors in the upper end of the femur should be approached surgically with caution. Irradiation is often to be preferred because of the technical difficulties of curettage in this location. Tumors in the fibula, ulna, ribs and metatarsals may be resected, if extensive. Otherwise they yield to curettage. In advanced cases resection and subsequent restoration of destroyed area by bone graft may be required. Tumors in other long bones are suitable for curettage. Especially so are those in the lower femur and upper tibia. Complete destruction by the tumor of the entire extremity of a long bone is generally a serious contraindication to curettage. Amputation should be reserved (a) for cases showing unquestioned evidence of malignant transformation, or (b) for cases so far advanced that conservative measures are obviously inadvisable, and finally (c) in the lower extremity where, after surgery or irradiation, the result is a limb definitely inferior functionally to an artificial one.

Recurrences after surgical treatment are usually due to imperfectly performed or poorly chosen procedures. Attention is seldom called to the failures following irradiation therapy. Such failures, however, are not uncommon. Serious late sequelae following irradiation exist, and their management is difficult and often unsatisfactory. Surgery is preferred for giant cell tumors in accessible locations because the results are more predictable, attained more rapidly and with better final function; because errors in diagnosis are avoided by providing the pathologist with all the tumor tissue; because undesirable irradiation changes in the soft parts and the adjacent joint are avoided. Promiscuous cutting into these tumors is strongly condemned. The entire tumor bearing area should be removed if any operation is done.

Pre- or postoperative irradiation seems to us to be of questionable value, and we feel it may be hazardous. It seems far better to decide upon the use of either surgery alone or irradiation alone, rather than to combine the two methods.

REFERENCE

[1] Coley, B. L., and Sharp, G. S.: Pathological Fractures in Primary Bone Tumors of the Extremities. Am. Jour. Surg., vol. 9, No. 2, pp. 251–263, August, 1930.

DISCUSSION.—DR. GEORGE E. BENNETT (Baltimore, Md.).—Giant cell tumor of the patella is one condition that can be treated with accuracy. If the tumor is a small one, it may be cured by curettage, but one should not hesitate to remove the patella after curettement in instances of large tumors or early recurrences as the end-result of such an operation is most satis-

factory. Murphy, in 1907, excised the patella and performed a plastic reconstruction with fascia lata, connecting the quadriceps and patella tendons. This operation, with its many modifications, gives one about 80 per cent of normal function in a knee joint.

Dr. William B. Coley (New York).—Dr. Bradley Coley has, I believe, presented a very timely and excellent plea for the surgical treatment of giant cell tumors of the long bones. There has been a growing tendency during the last 15 years to substitute irradiation for surgery in the treatment of these tumors. Regaud, of France, has long been an enthusiastic advocate of irradiation, and in a paper published by La Charité on the results obtained in Regaud's clinic at the Radium Institute, 100 per cent success is claimed. However, an analysis of the 14 cases reported by La Charité shows that 12 were giant cell tumors of the jaw, one of the spine, and only one of a long bone, the upper end of the humerus. Unfortunately, in the latter, irradiation was used in conjunction with resection. Therefore this series offers no help in trying to determine the value of irradiation in the treatment of giant cell tumors of the long bones.

The tendency toward irradiation was greatly increased in 1931, following a discussion of the treatment of giant cell tumors at the annual meeting of the American Roentgen Ray Society in Atlantic City. At that time the question was raised whether surgery should ever be employed, and in reply one of our most distinguished pathologists stated most emphatically that the surgeon should never be called upon to treat a giant cell tumor. He added: "I have seen so many bad results from recurrence, malignant transformation, infection of all types, acute and chronic, resulting in amputation, that I have no longer any room to file these cases in our museum."

That there have been some bad results following the surgical treatment of giant cell tumors no one can deny; but the main point to be considered is the frequency of these results and when they occurred. A careful survey will show that most of the bad results occurred two decades ago before the use of Dakin's fluid and at a time when the technic of curettage had been but little developed. With improvement of the latter, the bad results from surgery became fewer and fewer until during the last decade they have been practically eliminated in the hands of surgeons who have had a wide experience in dealing with these tumors. The most important step in the improved technic, in my opinion, is closure of the wound without packing or drainage after curettage and chemical cauterization. This method was introduced by Dr. Bradley L. Coley at the Memorial Hospital about ten years ago, and so far he has not had a single failure to secure primary union.

The only bad result following surgical treatment of giant cell tumor at the Memorial Hospital occurred in a case of my own, 20 years ago: a very large, rapidly growing tumor of the fibula in which a diagnosis of malignant tumor had been made from the clinical and roentgenologic evidence. A complete resection of the fibula was performed by Dr. J. P. Hoguet and myself. Not content with this, I very unwisely decided to have the large wound fulgurated with the Keating-Hart machine which had been recently brought over from Paris. The charred muscle offered a perfect soil for infection; three days later gas gangrene developed, and I had to perform an emergency amputation to save the patient's life. He made a good recovery and was well when last traced, ten years later. In this case the tumor was so extensive that most surgeons would have amputated in the first place.

In my earlier series, treated more than 20 years ago, in only three

cases was amputation necessary for an infected sinus following curettage; in two of these, the curettage had been done by another surgeon before the patient came to me. There is not a single case of sinus formation or infection following surgical treatment in the entire series treated by Dr. Bradley Coley and myself at the Memorial Hospital during the past 18 years.

The bad results following irradiation in the treatment of giant cell tumors are rarely mentioned and yet I believe they are more numerous and far more serious than the bad results following surgical treatment, even the surgery of a decade ago. Among those that I have personally observed are: severe burns causing prolonged disability and later intractable ulcers, which fail to heal and require amputation. In one case, a giant cell tumor of the upper end of the femur treated by irradiation in 1931, a very large burn developed which failed to heal after 18 plastic operations; the patient is now in a hopeless condition from ulceration and recurrence of the tumor. Again, in some instances irradiation has rendered the bones so brittle that fractures have resulted from slight strains, and the slow union of these fractures has meant prolonged disability. In two cases I have observed osteomyelitis develop later. In one case, a sarcoma of the upper end of the tibia which was regarded as cured by irradiation, osteomyelitis developed three years later, necessitating amputation. The other case, a giant cell tumor of the lower end of the femur, was frequently cited as one of the most brilliant cures of irradiation (180,000 mc.h. of radium had been applied in 1922 over a period of one year). Seven and one-half years later an extensive ulcer developed requiring prolonged hospitalization; eight and one-half years later osteomyelitis developed, which failed to respond to an extensive operation, and an amputation had to be performed the following year.

In my opinion there is greater danger of pathologic fracture developing after irradiation than after surgery. Our series shows that in 32 cases treated by irradiation, without biopsy, pathologic fracture occurred in nine; and in 22 cases treated by irradiation after biopsy or curettage, pathologic fracture occurred in nine also. While some of the bad results from irradiation occurred in the earlier years (1921, 1922, 1923), there has been little change in their relative frequency, some of the worst having occurred in the last five years. Phemister, of Chicago, has pointed out the unfavorable results of irradiation in the treatment of giant cell tumors of the femur, and our experience at the Memorial Hospital would seem to support this view. Of a group of 25 femur cases, 13 were treated by irradiation without biopsy with bad results in eight; and 12 were treated by irradiation after biopsy or curettage with bad results in four cases.

According to Bloodgood: "The greatest danger from irradiation is to persist in it too long, especially if the bone lesion is resectable, or to produce a deep-seated myositis which destroys the function of limb and makes amputation an operation of necessity. I have specimens of three limbs in the laboratory in which a bone was the seat of a benign cyst or osteitis fibrosa, and prolonged deep roentgen therapy had so destroyed the soft parts or necrosed the bone that amputation was made necessary. There are, in addition, five examples of giant cell tumors in which the introduction of radium into the bone shell, after curetting, was associated with an osteomyelitis which resulted ultimately in producing great deformity and loss of function, or making amputation imperative.

"These preventable results from irradiation have not all been in the hands of inexperienced radiotherapeutists. They have either been due to incorrect roentgenologic diagnosis which led to unnecessary irradiation for benign

tumors, or more frequently they were due to an attempt to cure malignant disease of bone irrespective of the results on the normal bone and soft parts. There is no necessity whatever for pushing irradiation treatment to this point."

I should like to make it clear that I am not opposed to the treatment of giant cell tumors by irradiation; in fact, one of the first cases reported in medical literature, in which a giant cell tumor was treated by the roentgen ray, was one of my own cases in which irradiation was used in conjunction with Coley's toxins. From the time the Bone Sarcoma Department was established at the Memorial Hospital, I agreed to permit all the service cases to be treated by primary irradiation. My own cases, and those at the Hospital for Ruptured and Crippled, I continued to treat by surgery. A comparison of these two groups, both numbering about 50 cases, in my opinion furnishes conclusive evidence of the superiority of surgical treatment for giant cell tumors of the long bones.

The greatest advantage of surgical treatment is that it furnishes one with a miscroscopic section of all parts of the tumor, and eliminates the error in diagnosis that occurs in at least one out of five cases treated by irradiation without biopsy—the method recommended by Ewing and Herendeen. Furthermore, it greatly lessens the period of disability, permitting the patient to return to work after a few weeks instead of the prolonged period (many months and sometimes two or three years) required for irradiation.

I agree with Ewing that surgery and irradiation should rarely if ever be combined in the treatment of giant cell tumors. While I have used irradiation in a few cases after curettage, the amount has always been very limited, not sufficient to seriously interfere with Nature's efforts to regenerate new bone to replace the portion destroyed by the tumor. Prolonged irradiation undoubtedly does lessen this process of repair.

Furthermore, and perhaps still more important, is the danger of prolonged irradiation after curettage transforming an originally benign giant cell tumor into a malignant sarcoma. In a recent paper on "Malignant Changes in the So-Called Benign Giant-cell Tumors," I reported 16 cases in which a benign giant cell tumor later became a malignant metastasizing sarcoma. Eight of these cases were treated by a combination of surgery (seven by curettage and one by open biopsy) and prolonged irradiation. This high proportion suggests a very strong probability that prolonged irradiation after curettage markedly increases the tendency to malignant transformation.

The Place of Coley's Toxins in the Treatment of Giant Cell Tumors.—The fact that giant cell tumors are so highly sensitive to Coley's toxins—far advanced inoperable tumors have disappeared under toxin treatment alone, and the patients have remained permanently well—makes it impossible to ignore the value of the toxins in any discussion of the treatment of giant cell tumors. On the other hand, I do not advocate the toxins as the method of choice in the treatment of operable giant cell tumors of the long bones, for the same reasons that I do not advocate irradiation. These are: (1) The impossibility of making a positive diagnosis of benign giant cell tumor from the clinical and roentgenologic evidence alone in at least one case out of five; and when the error is discovered it is usually too late to save the life of the patient by amputation; and (2) the greater time required to effect a cure compared with that required for surgical treatment. There remains, however, a very important field for the use of the toxins in giant cell sarcoma, and that is: (1) as a postoperative measure for one or two months after

curettage in the hope of lessening the number of recurrences. The latter occurred in 30 per cent of Bloodgood's series treated by surgery alone, and in 10 per cent of our own series treated by surgery and toxins; and (2) as the method of choice in the treatment of inoperable giant cell tumors, alone or in combination with irradiation.

DR. BRADLEY L. COLEY (closing).—The opinions expressed in the paper are those of my associate, Dr. Norman L. Higinbotham, and myself. We fully realize that there is room for a difference of opinion as to whether benign giant cell tumor of bone is better treated by irradiation or by surgery. From personal correspondence with both bone surgeons and roentgenologists we are led to believe that surgeons in general prefer surgical treatment for accessible lesions and irradiation for inaccessible ones, whereas roentgenologists tend to select irradiation for most cases regardless of location. Surgeons throughout the country hold surprisingly similar views as to the indications for irradiation or surgical treatment. Exceptions exist, however, and we should not, I believe, consider that the subject is a closed one. I would particularly emphasize our conviction that irradiation or surgery should be employed in a given case without mixing the two modalities, and further that preoperative irradiation is even more hazardous than postoperative irradiation. The interstitial use of radium or the introduction of radium capsules or tubes into a giant cell tumor that has been operated upon is unquestionably a hazardous procedure.

Two cases, to our knowledge, thus treated terminated as malignant giant cell tumors and caused the death of the patients, despite the fact that the original sections were regarded by the pathologists and the Bone Sarcoma Registry Committee of the American College of Surgeons as showing benign giant cell tumor.

I was interested in Doctor Bennett's report of the removal of a patella for giant cell tumor. This procedure seems wholly justified, although I have not as yet encountered a case occurring in this bone. Total removal of the patella was done in a case of synovioma of the knee, however, and we were gratified at the excellent function of the knee joint which resulted.

SOME OF THE HAZARDS OF IRRADIATION

JOHN STAIGE DAVIS, M.D.
BALTIMORE, MD.

IN THE vast literature on treatment by roentgen rays and radium, it is very noticeable that with comparatively few exceptions little, if any, emphasis has been laid on the untoward effects which frequently follow irradiation. As a matter of fact, in many of the articles such dangers are seldom even mentioned.

In several papers dealing with burns caused by roentgen rays or radium published during the last 15 years, I have endeavored to impress on those using these powerful agents for therapeutic purposes that there are certain very definite dangers to the patient unless the greatest care is exercised. Recently, however, a number of excellent reports have appeared which have really given facts that should be generally known instead of minimizing the untoward effects of irradiation, as seemed to be the unwritten law when writing on this subject.

In an excellent monograph published in 1934 by Colwell and Russ, chapters are given on the untoward results of irradiation in the skin, respiratory tract, circulatory system, alimentary tract, generative system, urinary tract, nervous system, eye, ear, bone, cartilage, muscle, connective tissue, adrenals, thyroid, thymus, mammary gland, radiation sickness, *etc.* These chapter headings will give an idea to those who are uninformed of the extent and wide distribution of the damage, which may be and has been done.

Inasmuch as there are still many who do not seem to realize the hazards of irradiation, I feel that it may not be amiss to again stress the importance of these dangers, and to reiterate that both radium and roentgen rays should be used for therapeutic purposes only by experts, and then with the utmost caution. In my work in reconstructive surgery, there have come under my care through the years a large number of patients who have had lesions which followed irradiation, which had been given for one cause or another. I have no personal knowledge of the technical application of roentgen rays or radium, but my experience with these agents has been entirely confined to the care of patients with lesions which have been caused by them.

No one can dispute the great value of roentgen rays and radium when applied for therapeutic purposes in suitable cases by those competent to use them, but the fact should be emphasized that frequently burns or other destructive tissue changes result, which in many instances are far more serious than the original lesion.

The enthusiasm of dermatologists and others who use radium and roentgen rays therapeutically is proverbial, and they regard irradiation as a panacea. The average user of radium and roentgen rays treats every growth, both

malignant and benign, with one of these agents, or with one and if this one is not effective, then with the other. It should be borne in mind that in many of these cases, a simple surgical excision is the method of choice, and if this method is used in suitable cases, a cure will be accomplished and the tissue changes which may be caused by irradiation can be avoided. Another fact which is often overlooked by the ray therapist is that some malignant growths are not ray sensitive and that instead of being destroyed, they are apparently stimulated in their growth by irradiation.

I am told by experts in roentgen therapy that, although the technic is constantly being improved, there is no excuse at the present time for a serious burn, if all the modern methods of dose measuring and machine calibration are properly carried out. When we add to this the other forms of protection for the patient and the operator now available, undesired tissue changes should become less and less frequent. However, I can still reiterate the statement made several years ago that I see more patients with ray changes each year. This increase in the number of ray burns is probably due to the fact that a larger group of patients are being irradiated than ever before.

I will consider briefly some of the untoward effects of irradiation and hope that the recital of these disasters may induce those who use these agents to be more careful in the application as well as in the selection of suitable cases for irradiation. My own experiences with these undesired effects have been principally confined to a large number of burns, which have been on every part of the body and to retardations of development. Many patients treated with roentgen rays or radium sooner or later after the acute reaction has subsided show the local characteristic signs of over irradiation: atrophy and dryness of the skin, loss of hair, pigmentation and telangiectasis, atrophy of the subcutaneous tissue, thickening and cracking of skin and nails, keratosis, ulceration and possible ultimate malignant degeneration. In the deeper tissues, fibrosis or destruction of muscles, necrosis of bone and cartilage, *etc.*

Most of the burns seen today are the result of the therapeutic use of roentgen rays or radium in dermatologic or other clinical conditions. Some follow the prolonged use of the roentgenoscope; some are due to over exposures by unskilled operators who often burn themselves as well as their patients; and some occur in spite of most careful exposures by skilled roentgen and radium therapists. There is also a fairly large group which follow depilatory treatment in beauty parlors.

Owing to the development of adequate protection for the operator, we seldom encounter serious ray changes on the hands of the modern trained roentgenologist, although we still see, now and then, on the hands of the pioneers in roentgenology and radiology, the blighting effect of multiple exposures to which they were subjected before the importance of protection was realized and real protection was available.

All degrees of burns may be produced from simple erythema, which follows the majority of treatments, to destruction of the whole thickness of the skin and varying depths of the underlying tissues. These burns may become

apparent very shortly after the treatment is given, but it is often found in the serious burns that the extent of the injury does not become apparent for months or even years after the treatments have been given. The reason for this long period of latency is well explained by Matas, who says, "The underlying pathology of these long delayed and necrogenic x-ray burns is a progressive proliferative endarteritis which obliterates the arterioles of the derm and subdermal connective tissue, leading to a gradual but fatal ischemia and nutritional disturbances in the radiated field." The same progressive proliferative endarteritis is found in delayed radium burns, and, to my mind, there is no other logical explanation for the delay in the appearance of tissue destruction in these ray burns.

Many of the burns which are referred to me are of long duration and have been treated by every conceivable local application. In many instances, roentgen ray burns have been treated with radium and radium burns treated with roentgen rays and various other rays. I cannot see the logic used in employing other destructive rays to restore tissue already completely changed by the original ray burn. My experience has been that when deep roentgen ray burns of long standing are treated with radium the condition is always made worse and vice versa.

When a deep burn has become definitely established, and local methods have been tried for a reasonable time without success, excision, with tissue shifting, is the only method which gives permanent relief from pain and which assures healing of the lesions as well as elimination of the danger of subsequent malignant degeneration. If the burn is small, it may be excised completely and the skin closed, or it may be removed by gradual partial excision.

Shortly after the discovery of roentgen rays, it was observed that living cells and growing tissues were markedly affected if exposed to these rays, and since that time considerable clinical and experimental investigation has been carried out along these lines. For years, I have observed that there is distinct retardation of the normal development of underlying bony structures in young children, in cases where new growths such as hemangiomata have been treated by irradiation, or, in other words, where the treatment has been given during the growing period. The retardation of the development of soft parts is also very evident in certain instances. I will outline an illustrative case.

When two or three months old, an infant with a hemangioma about the size of a five cent piece on the left side of the forehead was treated a number of times with radium. I first saw the child when it was two years old, and at that time the bony structure of the left half of the forehead was markedly undeveloped. There were definite ray changes of the soft parts, which in time will probably develop into a delayed burn. It is very doubtful whether this deformity caused by growth arrest will ever be naturally overcome, but it may be possible, when general development is complete, to fill out the retarded area by some plastic method. It was impossible to say at the time the patient

was seen whether there had been any damage done to the brain under this retarded area.

That the brain may be injured has been shown by several authors, and Markiewicz has recently reported a death with late roentgen lesions of the brain following roentgen ray radiation.

Another case of retarded development is that of a child who when three months old was treated several times with radium for hemangioma of the lower lip. She came under my care when 14 years old, after the completion of orthodontic measures. The lower lip was very small and thin, and was entirely composed of tissues which showed typical ray changes, both on the skin surface and inside the mouth. In addition, there was marked retardation of growth of the bone of the chin. Building out the contour of the chin and reconstruction of the lower lip was necessary in this case.

I have quite frequently seen the development of mammary glands in growing girls permanently arrested by irradiation used in treating thickened scars in the neighborhood of the breast. Localized retardation of development in growing children may occur in any region, or in any gland which is subjected to irradiation.

In a very interesting experimental study by Brooks and Hillstrom on the effect of roentgen rays on bone growth and bone regeneration, they found among other things that there was marked inhibition in the growth of bones exposed to a single massive dose of roentgen rays, and that the inhibition occurs only in the bone which is in the field of exposure.

In another group of experiments on young rabbits, they found that all animals receiving 40 per cent or more of the skin erythema dose showed definite inhibition of bone growth. Increased amount of irradiation always increased the amount of retardation. They found also that the total quantity of irradiation received always produced less inhibition of growth the larger number of times the dose was divided, or the longer the interval between the doses. Many more interesting facts were brought out in this paper, but the above will be sufficient for our purpose.

Facts such as these are either not known by many roentgen and radiotherapists, or if they are, are frequently ignored. My own feeling is that parents should be told of the possibility of such disasters, as retardation of growth as well as of ray burns, before irradiation is begun on young children. These untoward results may not be apparent for some time and the patient may never be aware of the primary cause of the growth arrest.

Much has been accomplished by deep roentgen ray therapy. Some years ago when this method of applying roentgen ray was in its infancy, I pointed out that it would be only a matter of time before burns of the overlying tissues and other disastrous deep tissue changes would develop in certain cases.

Some of the unfortunate end-results of deep therapy are now being reported in current journals, and it may be interesting to mention a few of them.

Only recently, Elliott and Jenkinson reported a death due to multiple

ulcerations of the stomach and small intestines following deep roentgen ray therapy applied to the abdominal region. I am convinced that serious consequences from deep roentgen ray therapy will be reported more and more frequently from now on, as more of this treatment is constantly being given and it is inevitable that bad end-results, directly due to tissue changes caused by the irradiation, will gradually come to light. In many of these patients treated by deep therapy, there are also marked changes of the superficial tissues, but these are seldom considered by the therapist of sufficient importance to mention, although they may give the patient continuous serious discomfort, and may also eventually undergo malignant degeneration.

The radium packs now being used for deep therapy must also be handled with the greatest care. I have seen severe burns follow the use of these packs, and have no doubt that in spite of all possible protection, deep tissue changes, which will have grave consequences for the patient, will in time also develop, as they have following deep roentgen therapy. Personally, I can see little advantage in any cure which while disposing of the original lesion leaves the patient in constant permanent torment.

In a recent paper, Huhner reports several cases where in spite of the greatest care in protecting the testicles, roentgen rays used in treating some dermatologic conditions of the thighs or perineum by experienced dermatologists interfered with the spermatogenic function of the testicle or destroyed it entirely. He believes that some testicles are very sensitive to the action of roentgen rays and that doses which are ordinarily considered harmless, in spite of what seems to be adequate protection, may cause sterility. The sterility of both males and females, who are constantly being exposed to roentgen rays in the course of their professional work, is well known.

Douglas Quick has shown that the use of radium in the treatment of vernal catarrh may be followed in some instances by serious consequences. Damage to the lens, as well as damage to the eyelashes, scarring and thickening of the lids and conjunctivitis have all been noted. These hazards should be thought of when radium or roentgen rays are used for therapeutic purposes on lesions in the region of the eye. Although these facts are of common knowledge, it is seldom that there is any hesitation on the part of the roentgen- and radiotherapist in using irradiation on lesions of the eyelids and adjacent soft tissues.

I have under my care at the present time a patient, 37 years old, who had been treated with roentgen rays for eczema of the face, off and on over a period of eight years, and was severely burned. In addition to several areas of beginning malignant degeneration in the burned area, one of which was on the eyelid, bilateral cataracts are developing, which undoubtedly have been caused by the ray treatment.

Nelson and Hirsch report a death due to late roentgen radiation necrosis of the larynx, which followed treatment of a lymph node of the neck. They review the literature and show that the injurious effect of irradiation on the larynx has been recognized for some time. In spite of this knowledge, this

therapy has been and is being used for the treatment of a varied group of affections of the throat and neck besides cancer, in what seems to me to be a haphazard way. The authors warn roentgen therapists of the dangers of damage to the larynx, which is apparently hypersensitive to irradiation, when irradiation is used in the treatment of carcinoma or other diseases of the neck, and stress the serious nature of such injuries to the patient. I have had under my care a number of extensive deep burns of the neck following irradiation of the thyroid, and in some of these a portion of the trachea had been destroyed.

I recently saw a woman upon whom a radical breast amputation had been done several years ago, following which postoperative roentgen radiation was given. This irradiation has caused almost complete cessation of thyroid function, and this cessation is apparently permanent. Finzi and others have reported serious changes in the lung following the treatment of carcinoma of the breast by roentgen rays.

Little is ever said before treatment is begun of the tremendous prostration, headache, nausea and vomiting, general discomfort, loss of appetite and depression, accompanied in many instances by blood changes, which often occur following both superficial and deep irradiation. These reactions are occasionally so severe that days may elapse before recuperation is complete, and there are cases reported where the outcome was fatal.

COMMENTS.—One has only to take care of burns and other tissue changes caused by irradiation to realize their great importance to the patient, as well as to those who produce these burns. Severe immediate and delayed burns sometime follow single exposures but are more frequently caused by multiple exposures. The susceptibility of the skin of certain individuals to irradiation must be taken into consideration.

The tendency to malignant degeneration in chronic roentgen ray or radium burns is interesting and important, and I see each year more patients in whom this degeneration has taken place. It has been noted that malignancy rarely develops in lesions resulting from a single massive dose or a few exposures at short intervals. It occurs more frequently in burns which are the result of many exposures over a long period of time.

When I read in the press that large and inexhaustible deposits of radium producing ore are reported to have been discovered, and that if this report is true, radium will be so cheap that it can be in the hands of most physicians, it makes me wonder whether it would be such a blessing after all, as I firmly believe that if this were ever possible, the harm done to patients by the ill-advised use of radium in incompetent hands would be many times greater than any benefit that could be obtained. The only reason, in my opinion, why there is any limit to the number of burns caused by radium is that it is still too expensive for general use.

At the present time, I understand that there are certain commercial companies who rent out radium itself or radium element (radon) to any member of the medical profession, who is able to pay the fee required. Doubtless this

dangerous agent is thus often put into the hands of well meaning but inexperienced persons, who, not realizing the danger, feel that if someone else can get results with radium, there is no reason why they cannot also. The ability to secure radium for therapeutic use is not confined alone to those renting the material, but sometimes radium can be borrowed from institutions and doctors or technicians in these institutions, who are without experience, can use it.

As far as I am able to ascertain, there is no control of the sale of roentgen ray machines or of radium in the United States or any other country. Any individual can purchase either of these highly dangerous agents if the money is available, regardless of his fitness to use them.

Much trouble would be avoided if the possible dangers, as well as the anticipated good results of irradiation, were explained to prospective patients, instead of subjecting them to this treatment without their being aware that danger exists.

I feel strongly *that all radium and all roentgen ray apparatus used for therapeutic purposes, both superficial and deep, should be so controlled that these agents could be in the hands only of those thoroughly competent and trained to use them.* I further believe that if the extent of the damage done by these two agents, as at present used, were realized, drastic regulations would follow. The sooner it does, the better for all concerned.

The instances of the hazards of irradiation used for therapeutic purposes mentioned in the preceding paragraphs, as you can realize, only touch the surface of such happenings. However, my hope is that they will be sufficient to make those who use these agents "stop, look, and listen."

REFERENCES

[1] Blair, V. P., Brown, J. B., and Hamm, W. G.: The Surgical Treatment of Post-radiation Keratosis. Radiology, vol. 19, p. 337, December, 1932.

[2] Brooks, B., and Hillstrom, H. T.: The Effect of Roentgen Rays on Bone Growth and Bone Regeneration. Am. Jour. Surg., vol. 20, No. 3, p. 599, June, 1933.

[3] Cilley, Earl I. L., Leddy, Eugene T., and Kirklin, B. R.: The Dangers of Roentgenoscopy and Methods of Protection Against Them. (I) General Review of the Problem. Am. Jour. Roentgenol. and Radium Therapy, vol. 32, No. 3, p. 360, September, 1934.

The Dangers of Roentgenoscopy and Methods of Protection Against Them. (II) Some Considerations of the Size of the Beam Used in Roentgenoscopic Examination. Am. Jour. Roentgenol. and Radium Therapy, vol. 32, No. 6, p. 805, January, 1935.

The Dangers of Roentgenoscopy and Methods of Protection Against Them. (III) The Protective Power of the Barium-filled Stomach. Am. Jour. Roentgenol. and Radium Therapy, vol. 33, No. 1, p. 88, January, 1935.

The Dangers of Roentgenoscopy and Methods of Protection Against Them. (IV) A Detailed Consideration of the Doses Received by the Fingers of the Examiner. Am. Jour. Roentgenol. and Radium Therapy, vol. 33, No. 3, p. 390, March, 1935.

The Dangers of Roentgenoscopy and Methods of Protection Against Them. (V) Some Considerations of the "Dose" Received During Examination of the Colon. Am. Jour. Roentgenol. and Radium Therapy, vol. 33, No. 6, p. 787, June, 1935.

The Dangers of Roentgenoscopy and Methods of Protection Against Them. (VI) Some Studies of the "Doses" Received by the Body of the Examiner. Am. Jour. Roentgenol. and Radium Therapy, vol. 34, No. 2, p. 241, August, 1935.

[4] Colwell, H. A.: The Method of Action of Radium and X-rays on Living Tissues. London, Oxford University Press, 1935.

[5] Colwell, H. A., and Russ, S.: Radium, X-rays, and the Living Cell. G. Bell & Sons, 2nd Ed., London, 1924.

X-ray and Radium Injuries. Oxford University Press, London, Humphrey Milford, 1934.

[6] David, Vernon C.: Radium Burns of the Rectum. ANNALS OF SURGERY, vol. 102, p. 422, September, 1935.

[7] Davis, J. S.: The Radical Treatment of X-ray Burns. ANNALS OF SURGERY, vol. 72, p. 225, August, 1920.

The Treatment of Deep Roentgen Ray Burns by Excision and Tissue Shifting. J.A.M.A., vol. 86, p. 1432, May 8, 1926.

Deep Roentgen Ray Burns. Am. Jour. Roentgenol. and Radium Therapy, vol. 26, p. 890, December, 1931.

Clinical Illustrations of Deep Roentgen Ray and Radium Burns. Am. Jour. Roentgenol. and Radium Therapy, vol. 29, p. 43, January, 1933.

[8] Elliott, H. R., and Jenkinson, E. L.: Ulcerations of the Stomach and Small Intestine Following Roentgen Therapy. Radiology, vol. 23, No. 2, p. 149, August, 1934.

[9] Finzi, N. S.: Late X-ray and Radium Effects. Brit. Jour. Roentgenol., p. 148, London, March, 1933.

[10] Gillies, H. D.: The Rôle of Plastic Surgery in Burns Due to Roentgen Rays and Radium. ANNALS OF SURGERY, vol. 101, p. 978, April, 1935.

[11] Gillies, H. D., and McIndoe, A. H.: Plastic Surgery in Chronic Radiodermatitis and Radionecrosis. Brit. Jour. Roentgenol., p. 132, London, March, 1933.

[12] Grier, G. W.: Radiation Therapy of Cancer of the Skin. Am. Jour. Roentgenol., vol. 32, p. 206, 1934.

[13] Huhner, M.: Sterility and X-rays. J.A.M.A., p. 1808, May 18, 1935.

[14] Lynch, M. G.: The Pathology of Radium Burns. Arch. Otolaryngol., vol. 21, p. 507, 1935.

[15] Markiewicz, T.: Über Spätschädigungen des memschlichen Gehirns durch Röntgenostrahlen. Ztschr. f. d. ges. neurol. u. Psychiat., vol. 152, p. 548, April 25, 1935.

[16] Matas, R.: Remarks on the Delayed or Remote Appearance of X-ray Burns after Long Periods of Latency. Am. Jour. Roentgenol., vol. 13, p. 37, January, 1925.

[17] Nelson, P. A., and Hirsch, E. F.: Roentgen Radiation Necrosis of Larynx and Other Structures of the Neck. J.A.M.A., p. 1576, May 4, 1935.

[18] Quick, D.: Radium in Vernal Catarrh. Arch. Ophth., vol. 4, p. 212, August, 1930.

[19] Rodick, J. C.: Roentgen Therapy in Non-malignant Conditions. New Orleans Med. and Surg. Jour., vol. 87, No. 12, p. 820, June, 1935.

[20] Scott, R. K.: Radionecrosis; A Clinical Study. Med. Jour. Australia, vol. 2, p. 1, 1934.

[21] Sittenfield, M. J.: Hazards in the Use of Radium in Cancer. Am. Jour. Roentgenol. and Radium Therapy, vol. 33, No. 6, p. 825, June, 1935.

[22] Stevens, R. H.: The Treatment of Radiation Injuries of the Skin. Radiology, vol. 19, p. 345, December, 1932.

[23] Taylor, L. S.: X-ray Protection. Am. Jour. Roentgenol. and Radium Therapy, p. 434, September, 1931.

[24] Wright, R. D.: Pathological Manifestations in Radionecrosis. Med. Jour. Australia vol. 2, p. 8, 1934.

DISCUSSION.—DR. JOHN A. MOORE (Houston, Tex.).—I think it is very fortunate indeed that Doctor Davis has called attention at this time to the dangers of roentgen rays and radium when used by persons who are uninformed and inexperienced in their use. I do not doubt that many of those here have seen ill effects in many cases from these agents. I regard these two agents as two of the most valuable that we have had given to us by science, when properly used. We are reminded also that there are many other agents that are being misused. In the Presidential Address of Doctor Payne he called attention to the situation we are facing constantly with ill trained surgeons, or with those without experience and training, who attempt to do things they are not fitted to do.

Hearing these papers, and hearing many discussions of other issues, I am reminded of a group of men who were talking with Will Rogers, when the Mayor came from the outer part of the gathering in an attempt to get Rogers to go to the platform and be seated. The Mayor said in a loud voice, to attract his attention, "I am Mayor Holcamp," and Will Rogers replied in his own way, "What are you going to do about it?" Unless someone goes preaching in the wilderness about these matters nothing will ever be done about it, but how many of us in the communities in which we reside are raising our voices in the correction of these various things? It seems to me that should be part of our attempt in the correction of these evils. I do not know what to propose, but as was intimated in a discussion yesterday, if this surgical group, or any group that is well trained, does not tackle these things in a corrective way, then I think our meetings are somewhat in vain, for it is only in such correction that we may ever hope to overcome these evils. Doctor Davis has called attention to the fact that the danger has increased now that radium has become so cheap, and that it is being rented to persons who have had no instruction in its use for as little as sixty dollars a month. For this sum you can rent 100 mg. of radium, and it is placed in hands from which much danger is sure to come.

DR. VILRAY P. BLAIR (St. Louis, Mo.).—Usually after Doctor Davis talks I could limit my comments to two words, "Me too." It would certainly be a great help if the use of roentgen ray machines and radium could be controlled as suggested, but we have tried prohibition! Of course, this paper of Doctor Davis's, and others like it, will gradually influence those who may influence others, but I think the direct attack at present is hopeless.

We have ourselves demonstrated the possibility of deep radiation damage in efforts to treat absolutely inoperable carcinomata by large doses, put in with the skin flaps retracted. Whether that is a good thing or not we are not yet sure, but we do know we have damaged the deeper structures by taking away the control of the skin. One good point is that these patients get little of the sickness they get when the dosage is put in through the skin.

There is no question that cancer is prone to develop later in skin damaged by radiation, but sometimes it is the original growth for which the roentgen therapy was given, some of the cells of which have remained entangled in the scar. I have seen that demonstrated 27 years after the control of a breast carcinoma of the glandular type. It recurred as a little nodule and we then removed that nodule and a large part of the scar, immediately covering the raw area with a free graft, almost immediately little nodules occurred all over the area from which we had removed the scar tissue.

DR. JOE V. MEIGS (Boston, Mass.).—Being in charge of radium administration for carcinoma of the cervix at the Massachusetts General Hospital and at the Massachusetts State Cancer Hospital at Pondville, I am greatly

interested in Doctor Davis's report, which I think is an extremely important one. The use of radium is not difficult to those who know nothing about it, but is most difficult, however, to those who do know something about it. I have seen over 1,000 cases of carcinoma of the cervix, and feel that I know something about what can happen to patients who have been treated with radium. There have been a number of cases of carcinoma of the cervix where the use of radium has produced a large fistula between the bladder and vagina. There are other patients who have died of kidney infection, in whom no evidence of carcinoma of the cervix could be found at autopsy, but in whom tremendous fibroses of the pelvis were present. I have seen one case recently where radium was given for uterine bleeding and within two years a peculiar carcinoma of the bladder occurred which appeared to be on the site of a radium change. Another patient treated by Doctor Dudley of Chicago 13 years ago for abnormal bleeding had a peculiar discharge for many years thereafter. She was a patient of Dr. W. P. Graves's for several years and came to me recently, Doctor Graves having died, and was found to have an unusual carcinoma of the body of the uterus with a metastasis to an ovary.

I would like to sound a warning to everyone to be careful and thoughtful in using radium, for disasters can occur and they are extremely unpleasant.

TUMORS OF THE KIDNEY

GEORGE R. LIVERMORE, M.D.
MEMPHIS, TENN.

TUMORS of the kidney may be malignant or benign. With the exception of cystic tumors, I agree with Bugbee and McKay,[1] who state that every tumor of the kidney should be considered malignant and treated as such. They may involve the parenchyma, the pelvis or both.

The classification of tumors of the kidney is a much mooted question. I shall not bore you with the various pros and cons of this controversy further than to state that I believe in the Grawitz theory of the adrenal rest origin of hypernephroma, that they vary in the grade of malignancy and that carcinoma and sarcoma both occur independently of hypernephroma and are probably due to intrinsic causes, *e.g.*, inflammation, toxemia, *etc.*, and to extrinsic causes, *e.g.*, trauma, stone, *etc.* The classification of tumors of the kidney by Bugbee and McKay with slight additions is as follows:

TUMORS OF INFANCY AND CHILDHOOD

Embryonic in Origin

Wilms' tumor (malignant)

TUMORS OF THE ADULT

Malignant
- (1) Parenchymal tumors. Hypernephroma. Grawitz
- (2) Adenocarcinoma
- (3) Papillary carcinoma
- (4) Sarcoma

Benign
- (1) Adenoma
- (2) Leiomyoma
- (3) Fibroma
- (4) Angioma
- (5) Lipoma
- (6) Lipomatosis
- (7) Solitary cysts

PELVIC TUMORS

(1) Benign? papilloma
(2) Malignant papilloma
(3) Squamous cell carcinoma

TUMORS OF THE CAPSULE

Lipoma

ETIOLOGY.—The cause of tumors of the kidney, like those in other portions of the body, is still purely conjectural. As previously stated, the Grawitz theory probably accounts for hypernephroma, while carcinoma and sarcoma are caused by trauma or irritation. Non-malignant tumors may be due to inflammation, toxemia or some metabolic disturbance. Reimann[11] divides hypernephroma into two types, typical and atypical. In the typical

type the cells are arranged in layers as in the cortex of the adrenal, while in the atypical the cells are irregular and not in layers. He states that the typical type is relatively benign while the atypical is definitely malignant. This, however, has not always been true in the cases seen by the author. The atypical, however, are more likely to be malignant, but many very malignant ones are of the typical type. Tumors of childhood are conceded to be of embryonic origin. It is true, too, that tumors having their origin in contiguous structures may invade and finally involve the entire kidney.

SYMPTOMS.—The great triad, hematuria, pain and tumor are the cardinal symptoms of tumor of the kidney. Unfortunately for diagnostic purposes, but fortunately for the patient, tumor is not always appreciable, for when it is palpable, it is usually in the late stage and the possibility of a cure is greatly diminished. Late symptoms, too, are anemia and loss of weight.

Symptomless hematuria is frequently the one and only sign of tumor of the kidney and it often requires the most thorough and painstaking urologic study to prove that a tumor is present. Sometimes small particles of tumor tissue may be voided in the urine, but this is a rare finding. Many other symptoms may occur in these cases that result from the tumor itself or from pressure or secondary infection, *e.g.*, there may be frequency, burning, tenesmus and nocturia, or the last may be present without the other three. Blockage of the ureter or a calix by pressure of the tumor or by blood clots, pus or a stone can cause renal colic. Dyspnea may be present and in some cases the blood pressure is low while in others it may be high. In the cases I have seen I do not feel that the tumor of the kidney played any large part in the production of high blood pressure. It is a fact, however, that toxemia, with resulting anemia and loss of weight, could easily account for a low blood pressure. This view coincides with that of Barney.[2] Fever, too, may be present but is due to infection, back pressure, absorption or metastases.

Varicocele is said to occur on the involved side. Although varicocele is of frequent occurrence in patients without tumor, I have never seen marked varicosities in a patient with a tumor of the kidney. George Smith[8] makes this same observation, but states that in Dr. Robin Pearses' Clinic in Toronto he saw a tremendous varicocele in a patient with a large left renal tumor.

Most cases occur in men. In the 28 cases tabulated, 20 were in males and eight in females (Table I). Barney,[2] however, reports 43 cases in females and 41 in males. Kretschmer,[3] in 1925, reported 40 males and 14 females and Foulds[4] reporting from the Mayo Clinic in 1923 found 72.42 per cent in males. George Smith and A. B. Shoemaker[8] report 62 cases from the Massachusetts General Hospital with 43 in males and 19 in females.

In childhood there are few symptoms. In fact, the condition is usually discovered only after the mother notices the abdomen increasing in size or a mass in the abdomen or loin. Blood and frequently pus cells will be found in the urine, although the passage of blood stained urine is rare. I would

TABLE I

SYNOPSIS OF TWENTY-EIGHT TUMORS OF THE KIDNEY

Case No.	Sex	Age	Race	Date of operation	Duration of symptoms	Tumors	Hematuria	Pain	Symptoms	Metastases	Results	Kidney involved	Character of tumor	Treatment other than nephrectomy
1	M.	47	W.	6-11-34	1½ yrs.	Yes	Yes	Yes	Suppression of urine. Loss of wt. Anemia	Left femur. Path. fracture	Died, October, 1934	Left	Hypernephroma	
2	M.	67	W.	4- 3-35	6 mos.	Yes, very large	Yes	Yes	Loss of wt. Dysuria, nocturia, weakness, anemia	No	Living, October, 1935. Weak, cachetic	Right	Hypernephroma	
3	F.	45	W.	4- 6-34	1 yr., 4 mos.	No	Yes	Yes	Frequency, dysuria, anemia	No	Living	Right	Epidermoid carcinoma of ureter and kidney, primary in ureter	
4	M.	?	W.	12- 4-34	13 mos.	Yes, large, nodular	No	Yes	Loss of wt. Anemia	No	Living	Right	Sarcoma	
5	F.	47	W.	No operation	2 yrs.	Yes	Yes	Yes	Anemia and loss of wt.	Left cervical nodes	Living	Left	Hypernephroma	Deep x-ray, 1934
6	M.	50	W.	No operation	1 yr.	Yes	Yes	Yes	Loss of wt. Anemia, dysuria	Left cervical nodes	Died	Left	Hypernephroma	Deep x-ray
7	F.	62	W.	10- 1-34	6 wks.	Yes	No	Yes	Loss of wt. Gastro-intes. anemia	Peritoneum, mesocolon, greater omentum, liver	Died	Left	Adenocarcinoma	Deep x-ray
8	M.	52	W.	No operation	?	Yes	No rec.	Yes	Cachetic, loss of appet. Loss of wt.	Lymph nodes, left axilla	Died	Left	?	Deep x-ray
9	F.	48	W.	No operation	6 mos.	Yes	Yes	Yes	Nausea. Loss of wt.	No	Living	Left	Hypernephroma	
10	F.	5	W.	April, 1935	5 mos.	Yes	Yes	No	Nausea, anemia, pain in abdomen	No	Died, 2 mos.	Left	Embryonic carcinoma	
11	M.	3	W.	No operation	2½ mos.	Yes, large	No	No	Nausea. Loss of appetite	No	?	Left	Embryonic (not operated)	

Annals of Surgery May, 1936

12	M.	50	W.	No operation	Neph. for yrs.	Yes	Yes	No	Suppression of urine. Anemia	No	Died	Right	Hypernephroma?
13	M.	53	W.	8- 2-30	1 yr.	Yes	Yes	Yes	Digestive disturb. Loss of wt. Rigors and Fever	Ribs	Died 3 years	Left	Hypernephroma
14	M.	58	W.	12-29-30	3 yrs.	Yes	Yes	Yes	Digestive disturbance	No	Died, 6 mos.	Left	Hypernephroma
15	M.	41	W.	3- 6-31	2 yrs.	Yes	Yes	Yes	No	No	Died, 1 yr.	Left	Hypernephroma
16	M.	2½	W.	9-14-33	3 mos.	Yes	No	No	Fever, gastro-intestinal	No	Died, 2 mos.	Left	Adenocarcinoma
17	M.	48	W.	8-25-34	4 yrs.	Yes	No	No	Digestive	No	?	Right	Hypernephroma
18	M.	46	W.	1-16-32	2 yrs.	Yes	No	Yes	Weakness, gastro-intestinal	No	?	Right	Hypernephroma
19	M.	56	W.	12-27-32	2 yrs.	No	Yes	Yes	Dysuria, had passed three stones last two years	No	Well, October, 1935	Right	Hypernephroma
20	F.	57	W.	4-16-32	3 mos.	No	No	Yes	Swelling of left thigh and leg	No	Died	Left	Adenocarcinoma
21	M.	58	W.	1-22-32	1 yr.	No	Yes, 6 wks.	No	Weakness, nocturia	No	?	Right	Hypernephroma
22	M.	7	W.	No operation	5 mos.	Yes	No	Yes	Frequency	Yes, lungs	Died	Left	Embryonic round and spindle cell sarcoma
23	M.	64	W.	1-21-32	3 wks.	No	Yes	No	Frequency, nocturia	No	Living and well	Right	Hypernephroma
24	M.	57	W.	6-14-33	3 yrs.	No	Yes	Yes	No	No	Living and well	Left	Hypernephroma
25	F.	59	W.	5-24-35	9 mos.	No	No	Yes	Frequency	No	Died, October, 1935	Left	Hypernephroma
26	M.	62	W.	7-31-34	6 mos.	No	Yes	Yes	Frequency and nocturia	No	Died, 1½ mos.	Left	Papillary adenocarcinoma of kidney pelvis
27	M.	45	W.	7-28-28	3 mos.	No	Yes	No	No	Yes, blad.	Living	Right	Papillary adenocarcinoma of renal pelvis
28	M.	45	W.	1930	3 mos.	Yes	Yes	Yes	Frequency, dysuria, fever and colic	Yes	Living	Left	Hypernephroma

therefore again stress the importance of the presence of pus and blood cells in the urine and urge you to insist upon a thorough urologic study to determine their source and cause.

As the tumor increases in size, pain and pressure symptoms such as ascites, edema of the external genitals and lower extremities and gastro-intestinal upsets occur. Rapid loss of weight and anemia are later manifestations, the latter often enhanced by hemorrhage within the tumor and unless it ruptures into the pelvis or calices does not appear in the urine.

METASTASES.—Metastases may occur, even in the early stages, to the lungs, pleura, liver, ribs, skull, scapula, long bones, peritoneum, omentum, renal vein and vena cava, lymph nodes of the neck and axilla, nose, cerebellum and pancreas. Even when these have occurred in hypernephroma, nephrectomy, followed by deep roentgen ray therapy, may give the patient many years of useful life.

DIAGNOSIS.—The early diagnosis of kidney tumors is often very difficult, even with the present day refinements in cystoscopy and roentgenology. The P.S.P. test in some cases is of little value, for even if the tumor is large, enough healthy kidney tissue may be left so that there is little reduction in output. It should be made routinely, however, as it may, with other evidence, be the deciding factor in the diagnosis. Plain roentgenograms and pyelograms, both retrograde and intravenous, should be made and repeated in order to check our findings and eliminate blood clots and spasm of the pelvis and calices, as well as fecal masses and distortion due to pressure of other structures. I agree with George Gilbert Smith[9] that intravenous urography is often unsatisfactory due to the diminished function in the involved kidney; though even when the function is good a satisfactory pyelogram may not be obtained. In children intravenous urography may be sufficient, but in many cases it does not indicate the functional capacity of the good kidney. I therefore advise retrograde as well as intravenous pyelograms; for cystoscopy can be done even in babies with the same degree of precision and with much less reaction than in the adult. I have performed cystoscopy and catheterization of the ureters of a girl two days old and a boy six weeks old.

TREATMENT.—All tumors of the kidney except the cystic types should be considered malignant or potentially so, hence nephrectomy should be done. When the tumor is very large and its removal would be too hazardous, preoperative deep roentgen therapy, as suggested by Walters,[6] and Bugbee and McKay[1] often results in such marked reduction in the size of the tumor as to render its removal much less difficult. It may also tend to prevent metastases by way of the lymphatics. Extraperitoneal removal is to be preferred and every precaution used to remove all malignant tissue and prevent contamination of the wound with tumor particles or infected material. It is exceedingly difficult in many cases to remove all the malignant tissue, for it is often found that the tumor has invaded the perirenal tissues by extension or by rupture of the capsule. If this has occurred or should the tumor

be ruptured during nephrectomy, flooding the wound with iodine followed by alcohol may prevent implants on the denuded areas. The perirenal fat should be completely removed. Transperitoneal nephrectomy is the operation of choice in the very large tumors that fail to reduce in size under deep roentgen therapy. The technic advocated by Wharton[5] makes it a less formidable operation. Eisendrath and others advise preliminary ligation of the vascular pedicle transperitoneally, to be followed later by extraperitoneal nephrectomy. If the patient's condition is poor and deep roentgen therapy fails to reduce the size of the tumor, I think this is a very excellent procedure.

Should nephrectomy be performed in the presence of metastases? This is a question of selection of cases. It is sometimes true that the patient is so uncomfortable from intercurrent affections such as infection, renal colic, hemorrhage, *etc.*, that regardless of his condition something must be done. It is also true that even when metastases have occurred the removal of the kidney, followed by deep roentgen therapy to both the kidney area and the metastases, sometimes gives the patient many years of useful life and freedom from pain. Bumpus[7] cites such a case with metastases in both lungs, living and well four years after nephrectomy. One of my own is similar to that of Bumpus. A man, aged 53, with an hypernephroma of the left kidney, had an intercurrent pyelonephritis and suffered frequent attacks of rigors, high fever and hemorrhages that could be controlled only by cystoscopy and the introduction of an indwelling catheter. The relief, however, was only temporary. There was roentgenologic evidence of metastases in the ribs. In view of the metastases we felt that nephrectomy would be useless, therefore advised deep roentgen therapy. As the attacks continued, the patient insisted that something be done. Nephrectomy was performed and later deep roentgen therapy was given. He made an excellent recovery from his operation, was free of symptoms for two years, but died of metastases in the lungs.

In tumors of the renal pelvis, the kidney, ureter and a section of the bladder about the ureteral orifice should be removed. If the patient's condition is too poor to permit this at one sitting, nephrectomy may be done and the ureter and its orifice removed later. Colston[10] has suggested removing the ureter, severing it near the bladder then destroying the mucosa of the remaining ureter for 1 cm. about its orifice with the high frequency current. If this proves as satisfactory as removal of the entire ureter and its orifice, it will obviate the necessity of the more extensive operation.

Coley's toxins have been recommended for malignant tumors of the kidney. Coley[12] reports four cases, a Wilms' tumor, an adenocarcinoma, an hypernephroma and a round cell sarcoma apparently cured and well after 29, 6, 16 and 23 years, respectively. I have had no experience with Coley's toxins in the treatment of tumors of the kidney and have seen no reports of its use except that of Doctor Coley.

CONCLUSIONS

(1) All kidney tumors except cysts should be considered malignant and treated as such.

(2) Nephrectomy should be done when possible as it may give relief from pain and several years of life even in the presence of metastases.

(3) Deep roentgen therapy may reduce the size of the tumor and make an inoperable case an operable one.

(4) In tumors of the renal pelvis the ureter and a section of the bladder about its orifice should be removed, or the ureteral orifice and adjacent bladder mucosa may be destroyed with the high frequency current.

REFERENCES

[1] Bugbee, H. G., and McKay, Robt. G.: Lewis Surgery, W. F. Frior Co., 1928.
[2] Barney, J. D.: Med. and Surg. Jour., February, 1913.
[3] Kretschmer, H. L.: Southern Med. Jour., February, 1925.
[4] Foulds, Gordon S.: Collected Papers Mayo Clinic, 1923.
[5] Wharton, L. R.: Surg., Gynec., and Obst., vol. 60, March, 1935.
[6] Waters, Chas. A.: Amer. Jour. Roent. and Radium Therapy, February, 1935.
[7] Bumpus, H. C.: Jour. Urol., vol. 20, No. 2, August, 1928.
[8] Smith, G. G., and Shoemaker, A. B.: Jour. Urol., vol. 14, No. 4, October, 1923.
[9] Smith, G. G.: Amer. Jour. Surg., vol. 30, No. 1, October, 1935.
[10] Colston, J. A. C.: Trans. Amer. G. U. Assn., 1933.
[11] Reimann, S. P.: Kidney in Health and Disease. Lea & Febiger, 1935.
[12] Coley, W. B.: Amer. Jour. Surg., vol. 29, No. 3, September, 1935.

DR. WILLIAM B. COLEY (New York, N. Y.).—Doctor Livermore has called attention to a case I originally reported before the American Urologic Society last winter, and I will give a few words of elucidation.

First, I wish to call attention to what you all appreciate, *i.e.*, the high malignancy of tumors of the kidney. The recent paper of Geschickter and Widenhorn on "Nephrogenic Tumors" (Am. Jour. Cancer, No. 4, December, 1934), showed almost 100 per cent mortality of kidney tumors in children and others as well; and the same was found to be true at the Presbyterian Hospital in Chicago on the service of Doctor Bevan. Of the four cases referred to by Doctor Livermore, only two were my own. It is important to know, however, that others, as well as myself, can get results with the toxins. It shows the two types of tumor in which I think Coley's toxins should be used by anyone who has to treat a tumor of the kidney.

In the cases that are inoperable, the toxins should be used as a prophylactic after operation. Two of the four cases referred to were inoperable. One of these was a woman aged 27, under the care of Dr. H. K. McDonald and Dr. A. S. Burns of Halifax, Nova Scotia. An exploratory operation revealed the lower pole of the kidney to be occupied by a large tumor which was adherent to a much larger growth anteriorly. A laparotomy was then performed disclosing a large retroperitoneal mass which it was impossible to remove. A section was removed and pronounced a round cell sarcoma by Dr. W. A. Lindsay. The patient was sent home to die. She had lost 40 pounds in weight. However, as a last resort it was decided to try Coley's toxins. The treatment was begun at once, and the patient showed immediate and rapid improvement, going on to a complete recovery. Fifteen years later, Doctor McDonald performed a hysterectomy for a fibroid tumor, at

which time he had an opportunity of examining the whole abdomen; there was no evidence whatever of the tumor of the kidney. She was well when last traced, 23 years later.

The second case treated by another surgeon was a woman aged 37, under the care of Mr. Arthur Connell, Professor of Surgery, University of Sheffield, England. An exploratory operation was performed, and a microscopic report of hypernephroma was made. The condition was inoperable. I advised the use of the toxins, which treatment was carried out by Mr. Connell. The patient made a complete recovery and was well 16 years later. This case was included in my report before the Third International Research Conference in Brussels in 1913.

The third case was a Wilms' tumor (adenosarcoma) in an infant aged 18 months. Dr. J. W. Perkins, of Kansas City, performed a nephrectomy for a large sarcoma of the kidney August 27, 1903. I advised using Coley's toxins as a prophylactic, and sent him a bottle that had been prepared by Dr. B. H. Buxton. The treatment was carried on for two years. This patient is in excellent health at the present time, more than 29 years later.

The last case was a male, aged 44, who was sent to me in December, 1928, by Dr. Charles G. Kerley, with a very large tumor of the kidney which had almost reached the inoperable stage. I referred him to Dr. Benjamin H. Barringer for a nephrectomy which it took two hours to perform. The patient was then given two exposures of high voltage roentgen therapy, after which the toxins were begun and carried out under my direction for three months by Dr. J. S. Chaffee, Sharon, Conn. This patient is still well at the present time, six years later.

If we can cure some inoperable malignant tumors of the kidney, and keep operable cases well for many years, with injections of Coley's toxins, I think the method is well worth trying.

ATRAUMATIC REMOVAL OF NEEDLE FROM HAND

UNDER FLUOROSCOPIC CONTROL

WILLIAM H. PRIOLEAU, M.D.

CHARLESTON, S. C.

THE difficulty of removing a needle from the hand often is not appreciated by those who have not attempted it. Accordingly it is frequently undertaken with inadequate facilities and by someone not sufficiently familiar with the surgical technic involved. As a result a common story is that a long search or several attempts have been necessary before the needle was located. The difficulty lies in the fact that during the operative procedure the needle moves quite readily among the ligaments of the hand, and thus the preoperative localization is of little help. The ligaments interfere with palpation and a bloody field makes recognition of the needle difficult, especially if it is discolored. Nerves and blood vessels are subject to injury, and irreparable damage may be done. The long search or the repeated attempts, with the resulting trauma, predispose to infection, which may cause death.

The technic about to be described was gradually developed following a conversation with Dr. B. H. Nichols, roentgenologist at the Cleveland Clinic. He advocated localization and fixation under fluoroscopic control followed by removal at open operation. In the proposed technic, the first principle is utilized but the second is fundamentally modified in that the necessity for the open operation has been practically circumvented. No claim to priority is made as this method is essentially an extension of the now generally accepted principle of localization and fixation under fluoroscopic control. However, a review of text-books and periodicals fails to show the technic here described.

While localization under the fluoroscope is generally accepted as being the only consistently satisfactory method, a number of technics for the extraction of the needle have been advocated. Carlos Santos,[3] of Lisbon, has been particularly interested in this subject and described an ingenious instrument with which he engaged the needle under fluoroscopic control and extracted it. It seems to have worked most satisfactorily in his hands. However, the objections may be raised that the instrument is not generally available, that it requires an unnecessary amount of manipulation under the fluoroscope, and that its withdrawal is made possible only by its cutting some tissues blindly. A number of electric localizers have been devised, but they have been generally found to have a limited application. Head fluoroscopes planned to give the operator intermittent fluoroscopic and normal vision have proven impractical. At present the most satisfactory method

Volume 103
Number 5

FIG. 1.

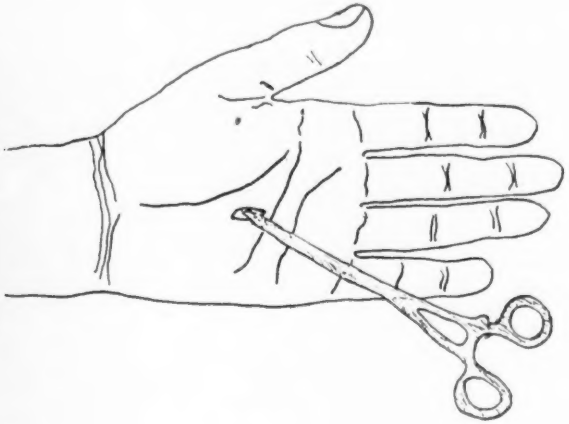
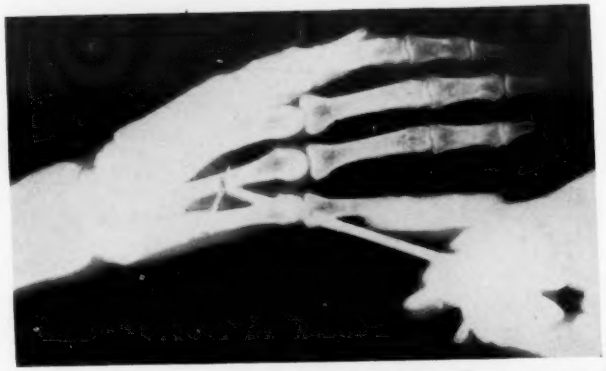


FIG. 2.

FIG. 3.



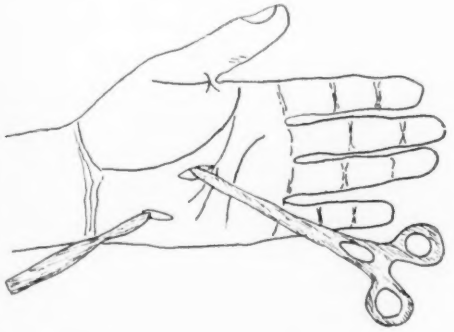


FIG. 4.

FIG. 1.—Roentgenogram of a needle in the hand.
FIG. 2.—Closed mosquito forceps in incision over needle.
FIG. 3.—Localization and grasping of the needle by mosquito forceps, under fluoroscopic control.
FIG. 4.—Second skin incision over the end of the needle, as determined by palpation.

used to any extent is localization and fixation under the fluoroscope followed by open operation.

The desideratum is to accomplish the removal without traumatizing the tissues, both on account of the danger of infection and of injury to important structures. This is done by localization and fixation under fluoroscopic control with removal by manipulation, of one end into a second skin incision. This reduces the trauma of operation to a negligible degree.

Operative Technic.—(1) After preliminary novocain infiltration, an incision 1 cm. in length is made through the skin over the needle, as located under the fluoroscope (Fig. 1). The point of a closed pair of mosquito forceps is inserted into the incision (Fig. 2).

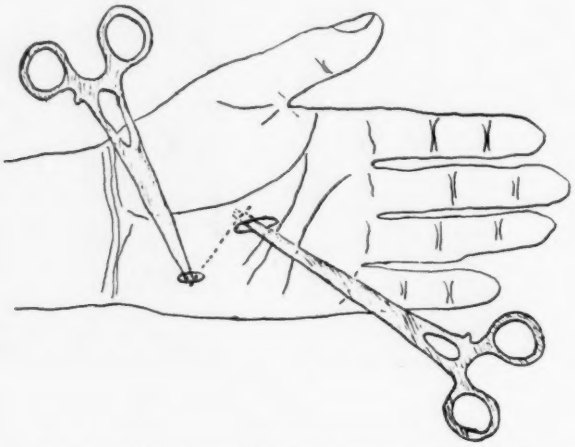


FIG. 5.—Needle being withdrawn through second incision.

(2) Under fluoroscopic control and by manipulation of the hand, the end of the closed forceps is advanced toward the needle. Once contact is established the forceps are opened slightly and made to grasp the needle, preferably near the middle (Fig. 3). The forceps are then securely locked, the fluoroscopic screen removed, and the light turned on.

(3) By manipulation of the forceps with one hand and gently pressing the surrounding skin with the index finger of the other hand, one end of the needle may be brought near the surface so that it can be palpated through the skin.

(4) Over the palpable end of the needle an incision ½ cm. long is made through the skin (Fig. 4).

(5) By further manipulation of the forceps and the index finger, the end of the needle can be brought into view in the second incision.

(6) This end is grasped with a second pair of forceps (Fig. 5); the first is unlocked; and the needle is easily withdrawn.

(7) Sutures are unnecessary as the edges readily approximate; also it is better that there be some provision for drainage.

(8) A roentgenologic examination is made for record and to determine if any part of the needle remains.

As regards the patient, the foregoing procedure consists of two small skin incisions and a little blunt dissection. Trauma is reduced to a minimum and accordingly minimizes the danger of injury and infection.

The mobilization of the needle utilized in this procedure is made possible by the same factors which ordinarily hinder its localization. It might seem as if the needle should be extracted by the first forceps, but this is not the case as there should be no unnecessary manipulation of the forceps under the fluoroscope, and it is easier to catch the needle in the middle than by an end. Also the forceps generally have some tissue within their grasp, which would prevent their withdrawal.

While the technic described is simple in principle, it is not equally so in practice. Locating and grasping the needle under the fluoroscope is difficult for those not accustomed to work in this medium. To a certain extent this is a blind maneuver but if the forceps are kept closed until in contact with the needle there is relatively little danger of doing any damage. There is some added difficulty in maintaining sterile technic under the fluoroscope. This is accomplished by adequate drapes; a sterile towel may be placed under the screen.

SUMMARY.—A technic is presented for the atraumatic removal of a needle from the hand. The needle is located and grasped with forceps under the fluoroscope. It is manipulated so that one end can be palpated under the skin. It is removed through a second incision.

REFERENCES

[1] Webb, R. C.: Foreign Bodies in Fingers. J.A.M.A., vol. 68, p. 1019, September 30, 1916.

[2] Bettman, R. B.: Removal of Needle with Intermittent Fluoroscopic Control. Surg. Clin. N. Amer., vol. 1, p. 1163, August, 1921.

[3] Santos, Carlos: Fortschritte auf dem Gebiete der Rontgenstrahlen, vol. 32, p. 369, 1924.

[4] Fitzsimmons, Duncan C. L.: A Scientific Method for Removing Metallic Foreign Bodies. Clin. Jour. (London), vol. 53, p. 594, December 10, 1924.

[5] Faggioli, R.: Extraction of Needle Deep in the Tissues. Semana Méd. (Buenos Aires), vol. 1, p. 571, March 11, 1926.

[6] Wright, V. W. Murray: Localization of Metallic Foreign Bodies by Electrical Means. J.A.M.A., vol. 90, p. 848, March 17, 1928.

[7] Goldblatt, D.: Removal of Foreign Bodies from Hands, Fingers and Feet. Am. Jour. Surg., vol. 8, p. 324, February, 1930.

[8] Bouyala, J.: Sur l'ablation des aiguilles dans la main. Gaz. Méd. de France, vol. 17, p. 484, October 1, 1930.

[9] Scollo, G.: Imbedded Needles. Policlinico (sez. prat.), vol. 39, p. 1223, August, 1932.

[10] Ganen, R., et Pérez, Albert: Collaboration—radio—chirurgicale indispensable pour l'extraction des aiguilles dans la main. Bull. et Mém. de la Soc. de Radiol. Méd. de France, vol. 21, p. 368, April, 1933.

[11] Henrard, Etienne: Deux cas de fragments d'aiguille de la main non justiciables de l'extraction sous écran. Bull. et Mém. de la Soc. de Radiol. Méd. de France, vol. 22, p. 215, April 10, 1934.

[12] Scavo, E.: Technique for Extraction of Needles from any Part of Body. Policlinico (sez. prat.), vol. 49, p. 816, May 28, 1934; p. 1570, October 8, 1934.

DISCUSSION.—DR. HARVEY B. STONE (Baltimore, Md.).—I have attempted to find needles in various parts of the body with no very great success. The hand is one of the most difficult places, and if Doctor Prioleau's method is successful I think he has brought out a very useful procedure.

DR. JAMES M. MASON (Birmingham, Ala.).—This is a rather unique and interesting procedure that Doctor Prioleau has reported. I had an interesting experience in the case of a piece of iron wire in the soft parts of the thumb. It gave him no trouble at the time of the accident but several weeks later the thumb became painful. A roentgenogram showed a good outline of the piece of wire in the ball of his thumb. I have manipulated a good many of these under the fluoroscope, but this seemed so simple that the fluoroscope was not used. I cut down and probed but never did find the wire. Another roentgenogram was made and there was no wire there. Through the process of oxidation or rusting, the wire had become entirely disintegrated, and the outline of the wire seen in the first roentgenogram was simply the iron rust which had remained in the tissues.

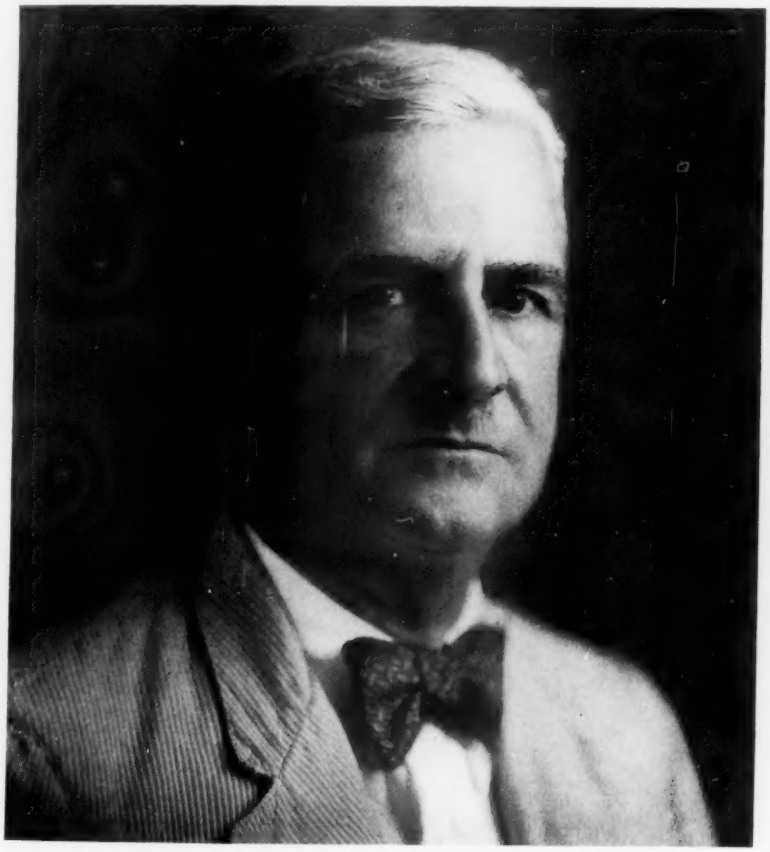
MEMOIRS

HERBERT PHALON COLE

1880–1935

HERBERT P. COLE, of Mobile, Ala., died while on a vacation in Miami, Fla., September 27, 1935, from a violent septic infection originating in a slight wound of a finger.

Coming at the height of his professional activity and usefulness, his death was a shock to his friends and a great loss to his community.



HERBERT PHALON COLE, M.D.

The Mobile *Register* said editorially, in part, concerning his passing: "The death of Dr. Herbert P. Cole in Miami yesterday deprives Mobile of the services of an eminent surgeon and a conscientious and useful public leader. In his nearly 30 years of residence in Mobile, Doctor Cole established for himself a distinguished reputation not only in medical circles, but in civic affairs. He was a citizen whom Mobile could ill afford to lose."

Doctor Cole was born in Minneapolis, Minn., September 2, 1880. He attended the high schools of Minneapolis and St. Paul, spent one year at the University of Minnesota, and was graduated from the University of St. Lawrence, at Canton, N. Y.

He studied medicine at Johns Hopkins University, and received his M.D. degree in 1906. Following his graduation, he served as intern in the Johns Hopkins Hospital in the department of gynecology, under Drs. Howard Kelly and Thomas Cullen. He went from there to the Lakeside Hospital in Cleveland, where he served as intern in surgery for a period of 18 months under Dr. George W. Crile. His work under these distinguished surgeons and teachers was at a time when they were doing their most brilliant and inspiring work, and their influence was reflected in his entire professional career.

After completing his hospital training, Doctor Cole located in Mobile and became connected with the School of Medicine of the University of Alabama as Associate Professor of Gynecology, and continued his activities as a teacher until the school was removed from Mobile to the University campus at Tuscaloosa.

Doctor Cole had a distinguished War record, serving with the rank of Major in Base Hospital No. 15, Fortieth Division, and, overseas, in Evacuation Hospitals 9 and 10.

In addition to his wide professional activities, he was much interested in civic affairs, and served a term as president of the Chamber of Commerce of Mobile.

He was a member of the County, State, and American Medical Associations, and of the American College of Surgeons. In 1928, he was President of the Mobile County Medical Society, and at the time of his death was attending surgeon to the Mobile Infirmary.

In 1909, he was elected to membership in the Southern Surgical Association, and was a frequent contributor to its programs and took an active part in discussions.

On September 14, 1909, Doctor Cole married Miss Jessie Wells, of Canton, N. Y., who, with two sons, survive him.

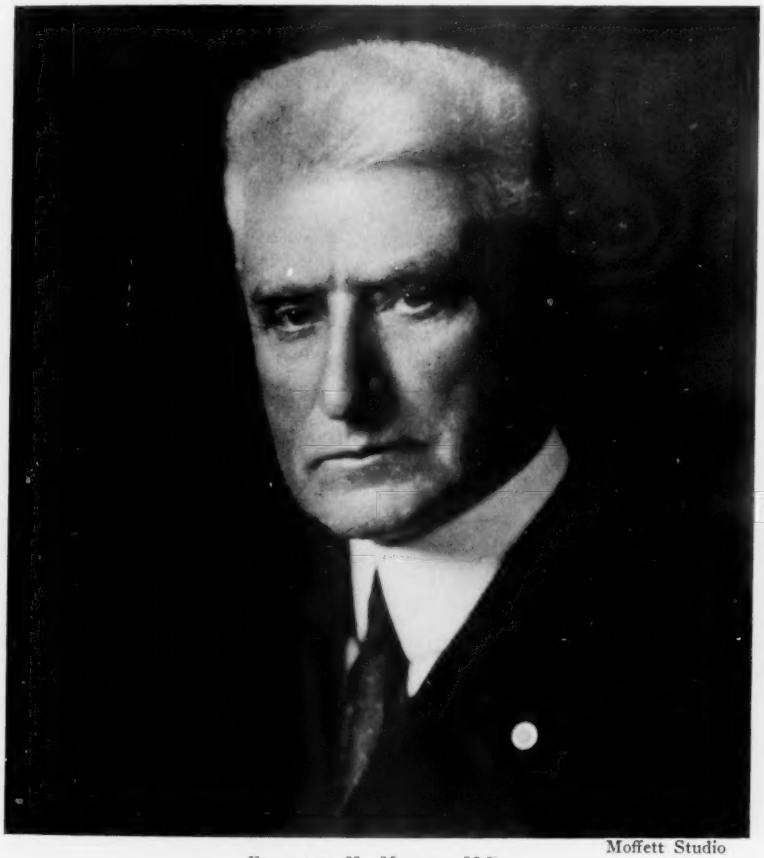
JAMES M. MASON

FRANKLIN H. MARTIN

1857–1935

"The nobler type of man is vexed at his want of ability, not because others do not appreciate him." *Confucius*, xv: 18.

THE life of Franklin H. Martin was characterized by courage. He was born with the hardy spirit of two pioneer families, each of which came west by caravans into the rugged wilderness of Wisconsin. The loss of his



Moffett Studio

FRANKLIN H. MARTIN, M.D.

father in the Civil War when he was five years of age; the remarriage of his mother and the happy union of two families of children; the struggles and sacrifices necessary to obtain his elementary education; his labors as farmhand, brickmaker, carpenter, janitor and, finally, school teacher are mute evidence of an indomitable courage to improve his abilities. To the man

so engaged from childhood in the attempt to increase his knowledge that its practical application might be of some benefit to his fellow men, there is little time left for vexation because others do not appreciate him. His early medical education is a recital of those exciting days which saw the advent of antiseptic surgery and the bacteriologic theories of medicine. His early medical career in Chicago was spent among men who were frank in discussion and whose chief attraction was fearlessness and their ability to make fast friends and implacable enemies, but to the latter they exhibited a spirit of fairness born of their own moral courage.

Early in his surgical career he became imbued with the idea of the need for dissemination of the rapidly increasing contributions to modern surgery. This thoughtful consideration of the actual workers in surgery who were distant from teaching centers, was the motivating factor of his life and will be recognized as the greatest contribution he made to surgical progress. The organizations for the elevation of the standards of surgery and the continuous education of all surgeons may change with the advancement of years but the courage to put into effect the basic principles of that idea, often against obstacles which would have deterred a less courageous man, showed a character devoted to the welfare of his fellowman. His Autobiography records these efforts in a manner typical of the untiring energy and industry of the man.

Fundamentally an idealist, he was extremely sensitive and was reluctant to approach people or speak in public to a degree that to many seems unbelievable. "Have the courage to do the thing you believe to be right but that you are afraid to do." This slogan, together with an ability to arrive at a logical conclusion with incredible speed, drove him forward with an impatience that to many gave the impression that his actions were often autocratic. Few men have been more misunderstood by those unwilling to withhold a quick judgment. From the very start there was no appeal for sympathy for the early hardships endured, no appeal for pity, but throughout only a steadfast, honest hewing to the line of what he considered to be his small contribution to the progress of his chosen profession. The final judgment of the surgical world will stand as his monument.

LOYAL DAVIS

EDITORIAL ADDRESS

Original typed manuscripts and illustrations submitted to this journal should be forwarded prepaid, at the author's risk, to the Chairman of the Editorial Board of the ANNALS OF SURGERY

Walter Estell Lee, M.D.
1833 Pine Street, Philadelphia, Pa.

Contributions in a foreign language when accepted will be translated and published in English.

Exchanges and Books for Review should be sent to James T. Pilcher, M.D., Managing Editor, 121 Gates Avenue, Brooklyn, N. Y.

Subscriptions, advertising and all business communications should be addressed

ANNALS OF SURGERY
227 South Sixth Street, Philadelphia, Pa.